

ATLAS OF
HISTOPATHOLOGY
OF THE SKIN

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ATLAS OF HISTOPATHOLOGY OF THE SKIN

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THIS WORK IS DEDICATED TO
SIR ROBERT McVITTIE GRANT
BART LL.D
TO WHOSE GENEROSITY IS DUE THE
FOUNDATION OF THE FIRST CHAIR OF
DERMATOLOGY IN GREAT BRITAIN

PREFACE

THIS work is presented in an attempt to portray in pictorial form the microscopical changes found in the more common diseases of the skin. For this reason the text matter has been severely limited just to what seemed necessary to explain briefly the processes illustrated. No references are given for these and for full discussions of the diseases mentioned, text-books of dermatology or original papers must be consulted. This is, therefore, a companion volume to such works and an introduction to the detailed study of the actual tissues in the laboratory. It is the result of co-operation between the dermatologist, the pathologist and the expert in laboratory technique and colour photography. The dermatologist sees the clinical lesion, its form and distribution; the pathologist sees the finer structure of the actual lesion and compares it with pathological processes as seen in other parts of the body or produced experimentally. Without each worker being in close touch with the other and each seeing the lesions from both points of view no undertaking is possible and little advance can be made in a true appreciation of cutaneous pathology.

The Atlas is intended primarily for post-graduate students, and especially those interested in the special study of Dermatology. It should serve as an introduction to the pathology of skin disease, and it is hoped it may stimulate some to pursue the subject by examining their own material. For the general pathologist, who often only sees occasional sections of skin lesions and these mostly tumours, it may serve as a useful reference.

The majority of the illustrations have been made from the authors' own specimens, but valuable material has also been placed at their disposal, and they wish to thank the following — Colonel W. G. Harvey I.M.S., Professor J. R. Leamonth, Professor Stuart McDonald, Junr.,

PREFACE

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Most of the material has been prepared and stained by Mr James Masson with the assistance of Mr James Paul of the Pathology Department of Edinburgh University and Mr Stanley Hay of the Laboratory of the Royal College of Physicians, Edinburgh, to whom our thanks are due for their skill and care.

The colour illustrations were made by the Finlay process, and accurately represent typical areas of the stained sections.

To the block makers we are particularly indebted for the infinite trouble they have taken over the many details and the obtaining of correct colour effects. Also we thank the printers, Messrs McLagan & Cumming Ltd. for the uniform excellence of their work.

Without the constant help and close collaboration of Mr Macmillan and Mr Parker of Messrs E. & S. Livingstone Ltd., our publishers, this Atlas could not have been produced.

THE AUTHORS

November 1946

CONTENTS

of	ix
INTRODUCTION	1
TERMINOLOGY	3
NORMAL HISTOLOGY	5
DYSTROPHY AND ALLIED CONDITIONS	35
INFLAMMATION	63
URTICARIA	64
LUPUS ERYTHEMATOSUS	75
PSORIASIS	79
PITYRIASIS ROSEA	85
LICHEN RUBER	89
PITYRIASIS RUBRA PILARIS	99
ECZEMA	103
CHEIROPOMPHOLYX	135
PRIMARY VESICULO BULLOUS ERUPTIONS OF INTERNAL TOXIC ORIGIN	139
PRIMARY VESICULO BULLOUS ERUPTIONS OF EXTERNAL ORIGIN	171
PUSTULAR ERUPTIONS	187
TINEA	199
ROSACEA AND RHINOPHYMA	203
GRANULOMA PYOGENICUM	204

CONTENTS

TRAUMATIC INJURIES AND THE EFFECTS OF STRESS

THE RETICULOSES

GRANULOMATOUS CONDITIONS

THE NAEVI

CYSTS

TUMOURS

INTRODUCTION

In arranging the various skin diseases, the histopathology of which is illustrated and described in the following pages, a clinical classification has been adopted, and they have been grouped according to the type of primary cutaneous change by which they are characterised and not according to their etiology known or presumed. This method has been employed for practical reasons, for skin diseases are much more individually distinct in their clinical aspect than in their pathology and the etiology is in so many cases obscure.

It is of great benefit to portray similar lesions in close proximity for purposes of comparison, because it is graduation of structural alteration which differentiates lesions which otherwise present many features in common both from the clinical and pathological standpoint. To arrange the diseases according to their etiology or the similarity of the course which they pursue, would be to lose this advantage of easy comparison, for a single etiological agent may evoke a variety of pathological changes according to the stage of the disease and the method in which it attacks the tissue. On the other hand, not to do so hampers the opportunity to trace the sequence of variations in tissue response in their continuity throughout the course of the disease. By cross reference however the obstacles presented by either method can be overcome, and it is hoped that while each one may be defective or open to criticism, the classification which has been chosen will prove the least inconvenient.

The histopathology of diseases of the skin can be studied with greater facility than that of any other organ and not only is it possible to correlate the clinical appearance of the eruption with the minute structural changes responsible for it, but the development progress, and regression of these changes can be examined as they proceed throughout the course of the disease. Although histopathology verifies the structural alterations which are suggested by the clinical appearance of the disease, it often happens that the clinical picture is much more striking and distinctive than that revealed by microscopic examination and it is not uncommon for eruptions which present widely different clinical appearances to show almost identical histopathological changes. In these circumstances it is the configuration of the lesions, their grouping, the surface area which they involve, their distribution on the body surface, their persistence, or constant repetition—all of which depend on the causative agent and on the biological state of the individual tissue cells, which give to the eruption its clinical identity and the histological changes offer no explanation for these features. It is therefore a matter for comment and reflection that a limited variety of pathological reactions and their resultant visible microscopic derangements in cutaneous structure can produce a much larger variety of visible clinical manifestations.

The distribution of eruptions which are of known or presumed internal origin presents a problem in itself and no explanation can be offered as to why certain areas of a single tissue should be selected to react with the noxious agent.

The microscopic differentiation of eruptions which are distinctive from the clinical standpoint may rest on minute and delicate findings, and in some cases may be impossible. On the other hand in a limited number of inflammatory diseases, and in the tumours, a precise diagnosis may only be possible when the histopathology of the lesion has been ascertained.

TERMINOLOGY

There are several terms used to indicate certain common abnormalities in epidermal structure and function which are peculiar to cutaneous pathology, and which therefore require to be defined.

ACANTHOSES. This refers to a general hypertrophy of the rete malpighi, which is accompanied by active mitosis in the cells of its deeper layers. There may be an associated cellular or intercellular oedema. The process usually involves the interpapillary cones of the rete which may be broadened and lengthened with a corresponding alteration in the papillae. As a result of this double alteration in the relations of the papillae and the rete cones it is not uncommon to find on section portions of the papillae cut on such a plane as to appear completely surrounded by epidermis, thus forming the meshes of an apparent network arrangement furnished by the epidermis. The stratum granulosum, lucidum, and corneum need not necessarily be hypertrophied in conjunction with the rete malpighii.

HYPERKERATOSIS This term indicates hypertrophy of the stratum corneum. The cells are normally keratinised and may form a solid mass or they may be loosely arranged to give a basketwork appearance. There is usually an accompanying increase in the layers of the stratum granulosum, but the underlying rete may be normal, acanthotic, or atrophic.

PARAKERATOSIS. In this state the process of keratinisation is upset and the cells fail to undergo the normal morphological and chemical changes as they pass from the rete to the stratum corneum. The stratum granulosum stage fails to develop and the cells reach the level of the stratum corneum in a somewhat swollen condition, imperfectly keratinised, and with their nuclei still present. In this condition they adhere to one another in clumps, which appear clinically as scales. This abnormal process is most commonly intermittent, so that the nucleated parakeratotic clumps of cells appear in layers betwixt cells which have undergone normal keratinisation. Oedema of the rete malpighii or its invasion by leucocytes are the usual visible antecedents and causes of the condition.

Special names have been applied to the varied appearances and effects of intercellular and intra-cellular oedema of the rete malpighii. Thus *Spongiosis* is the name given to marked intercellular oedema. *Ballooning* refers to intra-cellular oedema which causes gross swelling of the cell, and which may be accompanied by amitotic division of the nucleus. Fusion of adjacent cells takes place and results in the formation of large multinucleated cells which are referred to as balloon cells. Vacuolation with subsequent degeneration and disintegration of rete

cells is sometimes referred to as *alteration cavitaire*. *Dyskeratosis* is applied to the premature and abnormal keratinisation of individual cells or small groups of cells in the rete malpighii. These cells lose their prickles, become separated from their neighbours, the cytoplasm becomes strongly eosinophilic, and the nucleus pyknotic. On reaching the stratum corneum such cells usually retain their individuality the pyknotic nucleus persists or becomes fragmented, and they can be identified amongst the surrounding normally keratinised cells. The process affects the entire cell, and is quite distinct from the development of eosinophilic droplets in the cell cytoplasm which is not uncommonly found in isolated cells in a variety of inflammatory and acanthotic processes.

THE SKIN

NORMAL HISTOLOGY

This subject is dealt with in detail in various textbooks of histology and it is, therefore, unnecessary to describe minutely all the features of the normal skin. Here they are demonstrated by a series of illustrations, the descriptions of which will give sufficient detail for an understanding of and comparison with the pathological conditions which follow.

Normally the skin forms a waterproof and protective covering for the subjacent tissues and at the various orifices, nose, mouth, etc., it is continuous with the mucous membranes.

The skin consists of the epithelial layer or epidermis, well defined at its deep margin, and the dermis, or connective tissue layer. The dermo-epidermic junction is well defined, but the dermis is much less clearly demarcated on its deep surface, which merges with the subcutaneous tissue. The thickness and texture of both epidermis and dermis vary in different parts of the body according to local requirements, e.g. the skin of the sole of the foot is much thicker and denser than that of the body and there are also changes due to age, quite apart from disease.

Related to the skin are its various appendages—hairs, nails, sweat and sebaceous glands, whilst in the dermis are nerves and special nerve endings, blood and lymph vessels.

NORMAL SKIN—EPIDERMIS



FIG. 1

Skin from the sole of the foot—The epidermis is thick and has an especially deep stratum corneum, consisting of many layers of completely cornified epithelial cells from which all nuclei have disappeared and which consist largely of keratin. The layers on the surface are looser and more flaky (stratum dispartum). At the deep limit of the stratum corneum is, thus, pale line, the stratum lucidum, consisting of flattened cells filled with elastin and also devoid of nuclei. Immediately beneath this line is the granular layer or stratum granulosum, two to four cells deep. These cells are flattened, somewhat flattened and contain deeply stained blue granules of kerato-hyalin. Continuous with the stratum granulosum on its deep aspect is the rete malpighii, consisting of several layers of polygonal cells. These cells have well defined nuclei and between them are the intercellular spaces. The intercellular bridges ("prickles") which cross these spaces from cell to cell are not clearly seen except with higher magnification. The cells of the stratum granulosum, the deepest layer of the rete malpighii (rete mucosum), have columnar form and are known as basal cells. The lower limit of the epidermis is well shown and the downward projections ("rete pegs") of the malpighian layer are seen to alternate with the papillae of the dermis between them. The dermis consists of collagen fibres amongst which are seen several small capillary vessels. In the centre of the section there is duct of sweat gland. In the epidermis the duct appears as several oblique spaces in the cornified layer; in the rete beneath it appears again as spaces between the epithelial cells. There is no special lining to it as there are and its tortuous character is shown by its being cut in several places and not as straight tube. The gland from which this duct springs is not shown.

Hamann and Egan $\times 75$.



FIG. 2

Palm of hand—Thin glass section of skin mainly to illustrate the portico-like nature of the sweat glands and their ducts. The coil gland has deep in the dermis and its duct proceeds to the surface in somewhat convoluted fashion. A good example is seen to the left of the centre.

Frozen section Hematoxylin and Eosin $\times 15$



FIG. 3

Palm of hand—The epidermis has the usual layers and papillary form of its lower margin. Several sweat ducts are cut in different planes as they run to the surface from the sweat glands—some deeply placed in the dermis. There are also three sensory nerve endings—corpuscles of L. ann. Pacini—recognizable by their pale staining and concentric lamination.

Hematoxylin and Eosin 10

NORMAL SKIN—SWEAT GLANDS



FIG. 4

Sweat glands—This is a higher power view showing the ducts of the glands darker stained and of smaller caliber than the secretory coils. The latter have only a small lumen which is lined by cubical to columnar cells one layer deep. A thin basement membrane lies along the base of the cells and there is but a scanty supporting stroma around the coils. Just appearing in the field at the bottom is the outer margin of sensory nerve ending.

Hamshon and Essex $\times 95$

NORMAL SKIN—APOCRINE SWEAT GLANDS.

APOCRINE SWEAT GLANDS are modified types of sweat glands and are found in the axillae, nipples, pubic region and around the anus. In the female they have cycles of active secretion followed by resting periods corresponding to the menstrual cycle.



FIG. 5

Section of axillary skin—This shows hair coming to the surface and near its emergence is the duct of an apocrine sweat gland opening obliquely into the hair sheath. The apocrine gland itself is in the lower central part of the field.

Heddenham Anon Skin 35



FIG. 6

Apocrine sweat gland in its resting stage—The elements of the gland are larger and have wider lumens than those of the ordinary sweat gland.

Hematoxylin and Eosin $\times 100$



FIG. 7

High power of the same gland to show the type of epithelium—Closely applied to the base of the epithelial cells there are in places the elongated nuclei of the "micro-epithelial" cells—such by their contraction are concerned with the expulsion of the secretion.

Hematoxylin and Eosin 225

NORMAL SKIN—APOCRINE SWEAT GLANDS



FIG. 8

An apocrine sweat gland in its active phase—Compare with FIG. 6.
Hematoxylin and Eosin $\times 100$

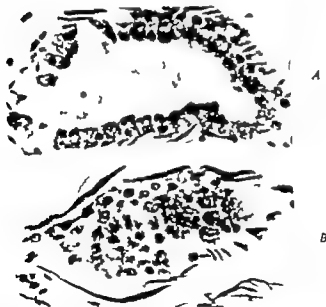


FIG. 9

A—The same under higher magnification to show the process of secretion—The inner zone of the cells becomes granular and disintegrates into the lumen to form the secretion.
 B—Another area of same showing the lumen filled with masses of granules and some cells. As the secretion is being pushed out, the cells are disintegrating. The dark layer represents all that is left of the partly disintegrated epithelial cells from these nucleated basal portions—new layer of cubical to columnar cells will develop (see next stage FIG. 7) *Eosin and Methylene Blue* $\times 400$



FIG. 10

Vertical section of normal scalp. — A low power view usually to show the disposition of the hair follicles. On the surface is the blue-stained epidermis resting on a relatively thick dermis continued as the deepest part as fatty tissue. Numerous hair follicles most of them cut obliquely are seen running up to the skin surface. At the base of each the bulbous growing end of the hair and each projects pear-shaped core of dermis which is continuous with the sheath of dermis around the epithelial sheath of the hair shaft.

Collodion section, Hamaker and Evans, x 25.

NORMAL SKIN—SEBACEOUS GLANDS

SEBACEOUS GLAND This opens into the hair follicle (from which it has arisen) about its upper third and is composed of cells of the rete which become loaded with fatty globules instead of becoming cornified. These fat laden cells disintegrate entirely (\Rightarrow holocrine type of gland) and so form the greasy sebaceous secretion. The glands open into the hair sheaths or on the skin surface by means of a duct lined by squamous epithelium.



FIG. 11

Opening of Sebaceous gland into hair follicle. A hair follicle, with portion of hair within it, has sebaceous-gland duct entering it at one side. On the opposite side is sebaceous gland having the usual characters but no duct is present in this plane.

Harshbarger and Evans $\times 100$



FIG. 12

Portion of sebaceous gland—At the periphery of the gland there is a thin narrow zone of flattened epithelial cells but as the centre is approached the greater numbers are transformed into large cells with central nuclei and vacuolated cytoplasm—vacuolated because the fat has been extracted from this section.

Hamann and Esner × 300



FIG. 11

Frozen section of sebaceous gland, stained for fat.—The lobules consist of solid masses of fat-laden cells, the fat being stained yellow—in the form of neutral fat and no doubly refractile lipid was demonstrable in this section.

Sabin III and Hamann × 100

NORMAL SKIN—NAIL.

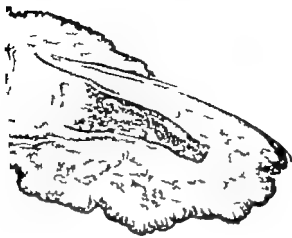


FIG. 14

Longitudinal section through nail on toe of rabbit—The clear area of the laminated nail plate is seen on the upper surface of the toe. Its free edge overlaps the epidermis—or hyponychium. Its proximal end is overlaid by the returned edge of epidermis—or epinychium. The base of the nail lying in the nail groove, reaches nearly to the phalanx and it is covered by its matrix, a thin layer of epithelial cells continuous with the invaginated rete malpighi. *Hammann and Egan* $\times 8\frac{1}{2}$

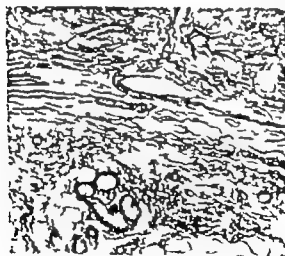
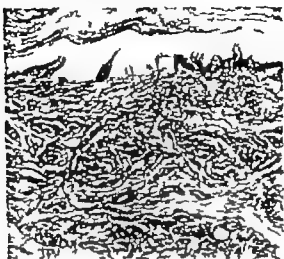


FIG. 15

Transverse section through lateral edge of nail—This is higher power than the preceding and shows the matrix of malpighian layer with which the nail plate continues. Overlapping the edge epidermis with considerable cornification. Underneath the nail plate is a thin even line of epithelial cells of malpighian layer. Beneath this the dermis in which are vessels and nerves seen on cross section. A small portion of phalanx seen below. *Hammann and Egan* 50

NORMAL SKIN—ELASTIC TISSUE.

ELASTIC TISSUE OF DERMIS. In addition to the collagen fibres of the dermis there are many elastic fibres distributed between the collagen and around hairs, sweat and sebaceous glands. The amount of elastic tissue varies in different regions and with age.



FIGS. 16 & 17

These sections are taken from the palm of the hand and show the whole thickness of the skin from the surface to the subcutaneous fatty layer. In Fig 16 there is the thick stratum corneum usual for this area, also thick and dense layer of collagenous fibres amongst which are seen delicate elastic strands, stained black.

In Fig 17 the deepest layer of the dermis is seen, with smaller elastic fibres merging with the subcutaneous fatty tissue as well as sweat gland. Elastic fibres can be seen surrounding the latter and also ramifying in the loose connective tissue.

Forchaff and L. M. Gann 75.

NORMAL SKIN—ELASTIC TISSUE.



FIG. 18

Skin from Abdomen.—The elastic tissue is relatively more abundant than that shown in Fig. 16. A hair and its sebaceous gland have delicate network of elastic fibres around them.

Wagner, Elastic Stain and Van Gieson. $\times 75$

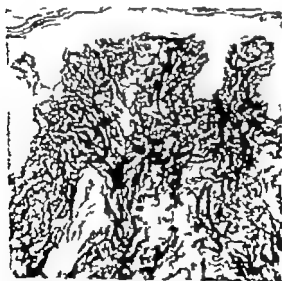


FIG. 19

Skin from Abdomen.—This is higher power view to show the numerous elastic fibres, their variation in thickness and their close relationship to the base of the epidermis.

Wagner, Elastic Stain and Van Gieson. $\times 400$.

NERVES. The skin is richly supplied with nerve fibres, medullated and non-medullated, and by certain special sensory nerve endings. Some of these fibres end on the epithelial cells of the epidermis, some round the hairs, sebaceous, and sweat glands, and still others form vasomotor fibres for the blood vessels. The special sensory organs are numerous about the fingers, hands and toes.



FIG. 20

Skin of palm aspect of finger. Meissner corpuscle—Situated in papilla of the dermis—an oval body composed of special cells around which are wrapped several nerve fibres—each are continuous with those leading away from the corpuscle.
Haddell. Silver Method and Camera. 260



FIG. 21

Skin of tip of finger. Vater-Pacini corpuscles ("Pacinian corpuscles").
—Several of these are seen, varying in size and shape, but all consisting of many concentric layers of laminated fibres in the centre of which is the ending of the nerve fibre (not specifically stained in this preparation).

Haemalum and Eosin $\times 45$.

DYSTROPHY AND ALLIED CONDITIONS

Both the epidermis and the dermis may be the seat of hypertrophy and degeneration, atrophy being in some cases the sequel to either of the other two phenomena. The conditions to be considered under this heading are ichthyosis and its variants, cutis laxa, elastosis senilis, and pseudoxanthoma elasticum, all of which may be regarded as physiological deviations—and amyloid infiltration, scleroderma, calcinosis cutis, dermatomyositis, granuloma annulare, and necrobiosis lipoidica—which are, on the other hand, essentially infiltrative or degenerative processes.

In **ICHTHYOSIS** (Figs. 22-24) the epidermis is congenitally atrophic, and its development is altered in such a way as to produce a hypertrophic stratum corneum which on its surface is subdivided by fissures into plates and facets. The associated under-development of the rete extends to the epidermal appendages, which are small and few in number.

In **CUTIS LAXA** (Fig. 25) the elastic tissue shows a regular hyperplasia throughout the dermis. **SENILE ELASTOSIS** (Fig. 26) and **PSEUDOXANTHOMA ELASTICUM** (Fig. 27) are also characterised by an increase in the amount of the elastic fibres, but in both conditions the fibres are irregularly thickened, whorled, and fragmented, and the process is not uniform but localised to portions of the dermis. There is little histological difference between a marked case of pseudoxanthoma elasticum and senile elastosis. Clinically however the former disease is usually distributed on the chest and flexures and occurs in young subjects, whereas senile elastosis affects exposed areas and is seen after the age of forty.

AMYLOID (Fig. 28) may occur in the skin as small localised deposits, or it may be diffusely distributed over large areas. In the first instance the deposit exists primarily and solely in the skin, produces an itchy lichenoid eruption, does not result from chronic sepsis in any of the internal organs, and is not accompanied by any deterioration in the general health. In the second instance it is part of a general amyloidosis dependent on chronic suppurative disease elsewhere in the body.

In the skin the amyloid occurs superficially in the dermis, first around the capillaries, and later as masses replacing the connective tissue fibres. There may be some leucocytic infiltration in the dermis, and the epidermis overlying localised deposits may be hyperkeratotic.

SCLERODERMA (Figs. 29 and 30).—In the early stages, the collagen fibres of the dermis are hypertrophied to form coarse homogeneous bands, there is considerable perivascular lymphocytic infiltration, and the deeper vessels show a diminution in the lumen due to proliferation of the intima and constriction by the swollen collagen. As the disease

progresses these hyperplastic changes gradually give place to atrophy with shrinkage of the skin, fibrosis of the dermis, loss of the papillae, and a thin epidermis.

CALCINOSIS CUTIS—The term calcinosis is restricted to the deposition of calcium in the skin with the formation of nodules and the condition is of rare occurrence. It may take the form of discrete nodules situated in the dermis—**CALCINOSIS CIRCUMSCRIPTA** (Figs. 31 and 32) or of an extensive general infiltration—**CALCINOSIS UNIVERSALIS**.

The circumscribed form may be associated with absorptive bone lesions, and, in these circumstances, has been described as metastatic calcinosis. In the absence of any preceding or associated impairment of bone formation the condition is regarded as being due to a disturbance of calcium metabolism.

The vicinity of the joints and tendons is the common site for the deposit and it is also sometimes seen in association with scleroderma. The condition has to be distinguished from the calcification which often occurs in sebaceous cysts and in areas of chronic inflammation.

DERMATOMYOSITIS (Figs. 33 and 34) presents a picture which suggests a moderate scleroderma as far as the dermis is concerned, but there is an almost complete absence of any infiltration of lymphocytes. The muscles are also affected by the degenerative process and show hyaline change and fragmentation of the individual fibres, with loss of their nuclei.

The clinical appearance of **NECROBIOSIS LIPOIDICA** (Figs. 38-42) and **GRANULOMA ANNULARE** (Figs. 35-37) may present a striking similarity and the structural alterations which are revealed by microscopic examination are almost identical. A large proportion of cases of necrobiosis lipoidica occur in diabetics, but the final distinguishing feature between the two diseases is the presence in the dermis of fatty degeneration or infiltration in necrobiosis lipoidica, and the absence of any such change in granuloma annulare. In both conditions there is degeneration and necrosis of the collagen and elastic fibres in circumscribed areas of the middle and lower dermis. The fibres are glassy in appearance and may show basophil staining, they are fragmented, and nuclear debris is scattered throughout the degenerating patch. Surrounding this area there is a fairly compact infiltration of lymphocytes and histiocytes, which have a palisade arrangement in granuloma annulare. It may be that in the early stages an infiltration of this type precedes the development of the collagen necrosis. In necrobiosis giant cells are not infrequently seen, and deposits of fat are present at the periphery of the necrotic area and to a less extent amongst the fragmented collagen fibres. In granuloma annulare localised areas of cellular infiltration similar to those seen in sarcoid have been noted in the vicinity of the necrotic focus.

ICHTHYOSIS

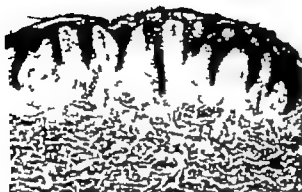


FIG. 22

Ichthyosis — The warts-papillary portions of the epidermis are narrowed, and the rete pegs are narrow and elongated. The cells of the rete appear compressed and smaller than normal, and the nuclei stain darkly. The stratum granulosum is absent and the ribbed stratum corneum rests directly on the rete. The collagen fibres in the papillary layer of the dermis appear to be homogeneous and stain faintly and in the deeper portion they are fragmented. Sebaceous glands are much reduced in number and are absent in this section. Sweat glands are also scanty and the cells of the ducts are flattened.

Harshman and Egan x 75

ICHTHYOSIS



FIG. 23

Ichthyosis Samolensis type—The stratum corneum is much hypertrophied. The rete is narrow, the stratum granulosum is absent, and the rete pegs are rudimentary. There is some perivascular lymphocyte infiltration. The glandular appendages are few in number.

Hansen and Emsen $\times 90$

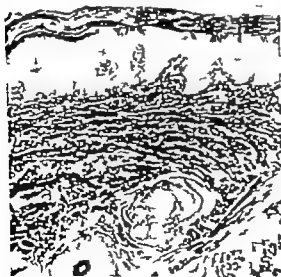


FIG. 24

Ichthyosis Samolensis type—The collagen and elastic fibres are fragmentary in places, some poorly, and in general are aggregated into various horizontal strands.

Isahoff and Van Gezen 100

CUTIS LAXA AND SENILE ELASTOSIS



FIG. 25

Cutis laxa — In this curious condition the skin is abnormally loose and can be stretched for many inches from its normal position, but it slips back when released. A vertical section shows great but regular increase of wavy elastic fibres throughout the dermis and extending into the subcutaneous tissue.

Wegert. *Elastic Stain and Tarcroft* $\times 40$

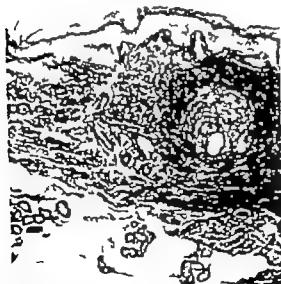


FIG. 26

Senile elastosis — The epidermis is thin. In the dermis, which is reduced in depth, there is relatively large amount of elastic tissue, irregularly distributed in clumps and layers and the collagen greatly reduced in amount.

Wegert. *Elastic Stain and Lill* $\times 75$

PSEUDOXANTHOMA ELASTICUM AND AMYLOID INFILTRATION



FIG. 27

Pseudo Xanthoma Elasticum—The epidermis is thinned and the rete pegs are absent. In the deeper layers of the dermis there are masses of thickened and fragmented bundles of elastic fibres. Nodules which stain poorly or are vacuolated may be seen here and there in the elastic fibres. No fatty infiltration is to be found in pseudo-xanthoma elasticum. The left-hand portion of the section shows that the increase and coarsening of the elastic fibres are not confined to the lower strata of the dermis. The collagen tissue and the skin appendages are normal. There may be slight degree of perivascular lymphocytic infiltration. The condition could be regarded as premature and localized example of senile elastosis with the essential difference that it is not associated with fatty degeneration in and amongst the affected elastic fibres. Verheeff and Van Gansen x 65.



FIG. 28

Amyloid infiltration—There is hyperkeratosis, but the essential feature is the violet-stained deposit occupying most of the dermal papillae, the collagenous fibres being apparently replaced by the amyloid. The vessels are seen running amongst the deposit, but their walls do not show any amyloid. Atthoff 1941 x 55.

SCLERODERMA.



FIG. 29

Scleroderma—The epidermis is thin and the rete pegs are absent. The dermis has homogenous appearance owing to the fact that the collagen fibres have become thickened and undergone hyaline change.

Henshaw and Egan $\times 50$.



FIG. 29a

Scleroderma—The same as Fig. 29 to show the thin epidermis and an increase of hyaline collagenous fibres amongst which are fragmented elastic fibres.

Lehrhoff and Leach $\times 75$.

SCLERODERMA.



FIGS 30 & 30A

Scleroderma.—The main feature is the very dense dermis, composed of hyaline collagenous fibres amongst which are fragmented, red like, elastic fibres—shown up black by the elastic stain in Fig 32

FIG. 30—Hamman and Essex 70

FIG. 30A—Lieberkühn and Van Gieson $\times 70$

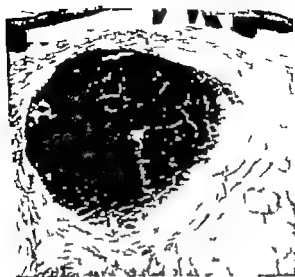


FIG. 31

Calcium Concentrate—Situated in the middle of the dermis there is a circumscribed deposit of calcium. The collagen fibres immediately surrounding it are abundant and are arranged in regular and concentric bundles; they are in direct contact with the mass. Farther away from the deposit the collagen has glassy appearance, probably due to pressure. There are no epidermal elements to be seen in the neighbourhood of the calcium, and there is no sign of inflammation. The epidermis overlying the mass is stretched. *Hammann and Enns* 55.



FIG. 32A

A—Medium power view of Fig. 31—This shows the point of contact between the collagen fibres and the mass of calcium.

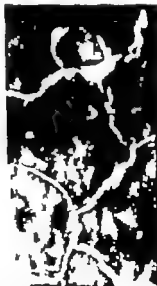


FIG. 32B

B—High power view of Fig. 31—Only calcium is shown in this illustration. It is in the form of an amorphous mass, throughout which are scattered small crystalline deposits.

FIG. 32 A. *Hammann and Enns* 130 FIG. 32 B. *Hammann and Enns* $\times 475$



FIG. 33

Dermatomyositis.—The epidermis is thickened and stretched, there is hyperkeratosis, and the rete pegs are absent. The collagen throughout stains poorly. Immediately under the epidermis it is atrophic, while in the deeper areas it is fragmented and there is edema between the bundles. There is no infiltration of inflammatory cells.

Harrison and Essex 60.

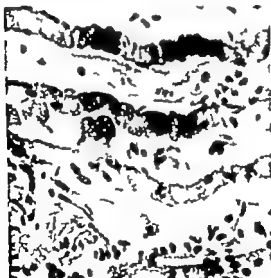


FIG. 34

Dermatomyositis.—The muscle fibres are atrophic and present varying stages of degeneration. The cytoplasm stains poorly and the nuclei of the sheath have undergone pyknosis. There is an infiltration of lymphocytes, and considerable edema between the muscle fibres.

Harrison and Essex 300.

GRANULOMA ANNULARE.

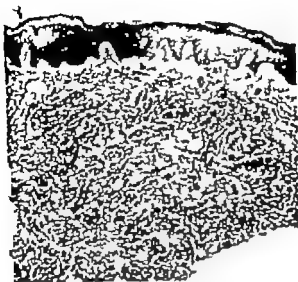


FIG. 35

Granuloma Annulare—Low power view showing glazy area of necrotic and fragmented collagen fibres surrounded by well marked cellular infiltration which shows tendency to be arranged in palisade formation.
Hamman and Essex $\times 50$.

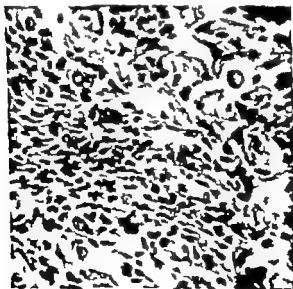


FIG. 36

Granuloma Annulare—This shows the infiltration surrounding the area of collagenous degeneration. The cells are small lymphocytes and histiocytes.
Hamman and Essex $\times 400$.

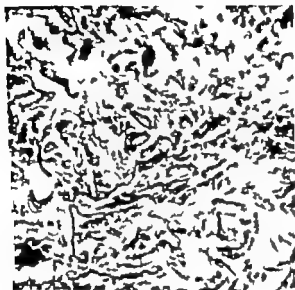


FIG. 37

Granuloma Annulare.—The collagen is fragmented and some poorly defined is present, and nuclear debris is scattered throughout the entire area.

Hamman and Egan $\times 220$

NECROBIOSIS LIPOIDICA.



FIG. 38

Necrobiosis Lipoidica.—There is a well defined strip of necrosis of collagen in the middle and lower dermis. The collagen has a glassy appearance, and nuclear debris is scattered throughout its substance. There is peri-vascular cellular infiltration in the vicinity of the area.

Hematoxylin and Eosin $\times 60$.

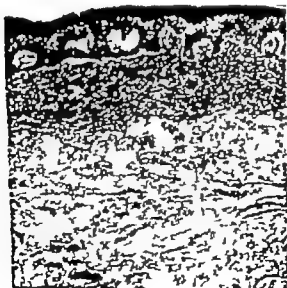


FIG. 39

Necrobiosis Lipoidica.—Similar to Fig. 38, but the necrotic area in the dermis is better defined by the method of staining.

Iron Hemateinyl and 1 on Gram $\times 60$

NECROBIOSIS LIPOIDICA.



FIG. 40

Necrobiosis Lipoidica.—This is a portion of the same lesion shown in Fig. 38. Numerous fat droplets are lying free amongst the degenerated collagen fibres.
Safran III and Haematexylin $\times 55$

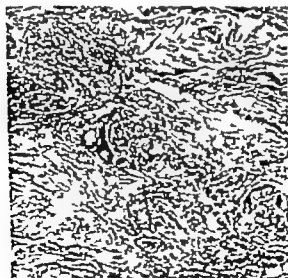


FIG. 41

Necrobiosis Lipoidica.—This shows an area of necrotic collagen and surrounding infiltration of small round cells. Several "foreign body" giant cells are to be seen at the centre of the section.
Haemalum and Eosin 110.

NECROBIOSIS LIPOIDICA.



FIG. 42

Necrobiosis Lipoidica.—Above, there is an area of fragmented and degenerated collagen containing nuclear debris. Below the collagen is swollen, cellular structure preserved, and there is an infiltration of lymphocytes and histiocytes.

Hematoxylin and Eosin $\times 350$

INFLAMMATION

Inflammation of the skin can be caused by an infinite variety of noxious agents which may reach its tissues either from without or from within, via the blood stream. In some cases the causative agent is a universal irritant in others its action is only exerted under special conditions of cutaneous predisposition or idiosyncrasy. With regard to the former group of irritants, individual skins show a varying susceptibility to their action, some being hyposensitive and some hypersensitive when compared with the average cutaneous sensitivity. The intensity of the response depends on three factors, the concentration of the irritant, the degree of cutaneous sensitivity of the individual, and the lack of immunity to their action.

In the case of the second group of substances the development of an inflammatory cutaneous reaction depends entirely on the state of the individual skin, the concentration of the irritant being of little importance. In the absence of a suitable state of cutaneous reactivity to which the terms idiosyncrasy and allergy are applied, the tissue is immune to the effects of the agent.

From the standpoint of histopathology inflammation of the skin is only significant when the vaso-dilatation and oedema is accompanied by visible structural or histo-chemical alterations in one or other of the component parts of the skin, or when the cellular reaction which may accompany the vasomotor phenomena presents distinctive characters. It therefore follows that the histological changes found in many of those transient erythematous eruptions, which present a striking and easily distinguishable clinical picture, are merely those of simple inflammation in which special distinctive features as an aid to differential diagnosis are entirely lacking. No individual mention of these eruptions seems necessary in an atlas on histopathology since their microscopic appearance sheds no light on the nature of the process by which they evolve or the causative agents by which they are produced, and the conception of such conditions must meantime be based solely on clinical data. Even in those inflammatory diseases in which distinctive histological changes are found it may be necessary to take clinical facts into account in order to assess the significance of the histological findings and elucidate the method by which they have been produced.

URTICARIA.

There are three clinical types of Urticaria, namely the common variety designated Urticaria, which is characterised by the transient appearance of wheals. Urticaria Papulosa, or Strophulus, which occurs in children and in which the lesions of ordinary urticaria become transformed into papulo-vesicles which erupt in crops and persist for a few days. Urticaria Pigmentosa, a rare condition in which the wheals are of a more permanent character than in the two foregoing types, and which is associated with pigmentary changes which persist indefinitely.

The clinical lesions of ordinary URTICARIA (Figs. 43 and 44) are striking, but no structural change accompanies them, as they are due entirely to a transient vasomotor reaction involving a dilatation and an increase in permeability of the minute vessels of the papillary bodies, and an arteriolar dilatation. Separation of the collagen fibres by oedematous fluid and perhaps some swelling of the fibres themselves, and stretching of the epidermis is all that is noted histologically. There is no cellular reaction.

In URTICARIA PAPULOSA (Figs. 45 and 46) the epidermis shows localised areas of intercellular and intra-cellular oedema, with vacuolation of individual cells in the rete, some of which stain poorly and show pyknosis. The stratum corneum may be thickened and acanthosis with broadening of the rete pegs may be present. There is a decided perivascular infiltration of lymphocytes surrounding the vessels in the superficial vascular plexus.

Urticaria results from the action on the skin of some substance to which the tissue is allergic, the localisation of the allergic process, and hence the clinical type of the reaction, being different from that in eczema. The noxious substance may reach the allergic cells either through the blood stream or as a result of intra-cutaneous inoculation from the outside, such as may occur from the action of stinging plants or biting insects.

Urticaria Papulosa is presumed to be an allergic reaction dependent on some unknown toxic agent of internal origin. It should be noted, however that the bites of FLEAS (Figs 49-51) and BUGS (Fig 52) may cause lesions which are identical with those of urticaria papulosa as regards their individual clinical features, but which are differentiated from it by considerations of distribution and course.

In URTICARIA PIGMENTOSA (Figs 47 and 48) there is an increase of melanin in the cells of the basal layer and a varying degree of perivascular lymphocytic infiltration. In addition to lymphocytes, histiocytes and plasma cells are scattered throughout the upper dermis, and a varying number of basophil or mast cells are present, the latter constituting the main diagnostic feature of the lesion. In children, a dense infiltration of mast cells is a characteristic and prominent microscopic feature.

URTICARIA.

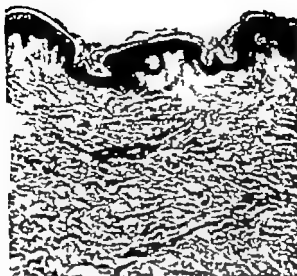


FIG. 43

Urticaria—The epidermis is stretched but is otherwise normal. There is no vasodilatation to be seen, the vessel having collapsed as a result of the rise in the surrounding tissue pressure consequent on the edema. The collagen fibres are separated by the capillary fibrils.

Hamaker and Egan $\times 70$.



FIG. 44

Urticaria—The collagen bundles are separated by edema and present fine reticular structure. The fibrils themselves are swollen and stain poorly.

Hamaker and Egan 420



FIG. 45

Urticaria Papulosa.—The stratum corneum is thickened and the rete is the seat of inter cellular and intra-cellular edema. There is moderate acanthosis, with broadening of the rete pegs. A well marked rim of lymphocytes surrounds the vessels in the superficial dermis. The collagen bundles are somewhat swollen and separated by edema.

Herman and Egan 80.



FIG. 46

Urticaria Papulosa.—The stratum corneum is thickened. The rete is acanthotic and arborescent, the arbores being both intra-cellular causing vacuolation, and intercellular. The arborescent rete cells stain poorly and the nuclei are pyknotic. There is lymphocytic invasion of the epidermis, and well marked perivascular lymphocytic infiltration surrounding the vessels in the dermis. *Herman and Egan* 140

URTICARIA PIGMENTOSA.



FIG. 47

Urticaria Pigmentosa. Adult type—There is an increase in the melanin content of the basal layer. In the dermis the blood vessels and lymph spaces are dilated, and lymphocytes, plasma cells, histiocytes and basophil or mast cells are scattered throughout it.
Giemsa stain $\times 100$

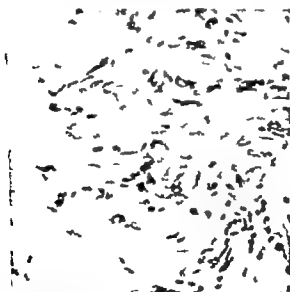


FIG. 48

Urticaria Pigmentosa—The vessels are dilated and lymphocytes and few mast cells are scattered throughout slightly indurated dermis. The section is from lesion in an adult. In children mast cells are much more in evidence.
Giemsa stain 300

FLEAS

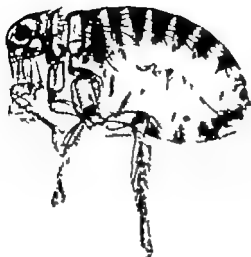


FIG. 49
The Common Flea—*Pulex irritans*. Adult female.
Cleared Preparation $\times 18$.



FIG. 50
The Common Flea—*Pulex irritans*. Adult male.
Cleared Preparation 18.

RAT FLEA AND BED BUG



FIG. 51
The Rat Flea—*Xenopsylla cheopis*. Adult female.
Cleared Preparation $\times 25$



FIG. 52
Bed Bug—*Cimex lectularius*. An adult male as seen
from the dorsal aspect.—The parasite has mahogany
colour as shown.
Cleared Preparation 8

LUPUS ERYTHEMATOSUS

(Fig. 53)

This condition is characterised histologically by a chronic inflammatory reaction in the dermis associated with degeneration of the collagen and elastic fibres, and secondary atrophic changes in the epidermis.

The vessels of the papillary layer of the dermis are dilated, and there is a sparse perivascular lymphocytic infiltration. In the middle and lower portions of the dermis this infiltration is more dense and forms clumps of cells surrounding the vessels, the sweat glands, and, to a less extent, the sebaceous glands. The elastic and collagenous fibres in the infiltrated area and its immediate surroundings are fragmented and degenerated.

The epidermal changes appear to be secondary to this low grade inflammatory reaction. They consist of thinning and stretching of the epidermis with loss of the rete pegs, and the appearance of hyperkeratosis which forms horny plugs in the mouths of the hair follicles.

The disease is generally regarded as having a definite connection with tuberculosis, and has affinities with the sarcoids and granuloma annulare. Tubercle bacilli and a tuberculous type of cellular reaction are however absent in lupus erythematosus.

LUPUS ERYTHEMATOSUS



FIG. 33

Lupus Erythematosus.—The epidermis is stretched and atrophied, and the rete pegs have more or less disappeared. There is hyperkeratosis and the follicles are plugged by masses of keratin. The papillary vessels are dilated, and there is perivascular lymphocytic infiltration which is scanty in the superficial dermis but dense in the lower and middle portions. The collagen and elastic fibers are destroyed in these lymphocytic clumps.

Hamman and Essex, × 60

PSORIASIS

(Figs 54-57)

The most striking feature in *psoriasis* is the development of an extreme degree of parakeratosis, the flattened and nucleated surface cells adhering together to form a succession of layers which replace the stratum corneum. Leucocytes are to be seen scattered throughout the parakeratotic cells both singly and in clumps, the latter resembling minute abscesses. The subjacent stratum granulosum has ceased to exist in many places. The rete is much hypertrophied and there is a pronounced elongation of the inter-papillary processes. The suprapapillary areas are thin, however and the tips of the papillae are separated from the parakeratotic surface only by a few flattened cells (Fig. 55). Here and there polymorphonuclear leucocytes can be seen in the intercellular spaces of the rete as they wander upwards towards the surface. Some of the rete cells are hydropic, but throughout the epidermis as a whole there is little or no oedema. The papillae may be slightly oedematous, and the vessels are dilated and surrounded by a scanty lymphocytic infiltration. There is nothing to indicate why the extreme and persistent alteration in the process of keratinisation should take place.

PSORIASIS



FIG. 54

Psoriasis.—General view of psoriasis lesion showing the heaped up layered scale containing collections of polymorphs, absence of the stratum granulosum, acanthosis, perivascular lymphocytic infiltration surrounding the terminal capillary loops and the upper arteriole plane.

Hemmelen and Emsw X 55



FIG. 55

Psoriasis.—This shows parakeratosis, absence of the stratum granulosum, acanthosis, elongation of the rete pegs, narrowing of the upper-papillary portions of the rete, vascular dilatation of small vessels in the papillae surrounded by lymphocytic infiltration and some colloid.

Hemmelen and Emsw 75

PSORIASIS

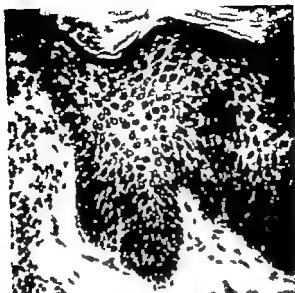


FIG. 56

Psoriasis—There is irregularity of the stromal granuloma which is absent in places; an acanthotic rete in which the cells are to all appearances normal. Few leucocytes wandering through the rete, edema of papilla, with vascular dilation and perivascular lymphocytic infiltration.

Henshaw and Egan $\times 200$.

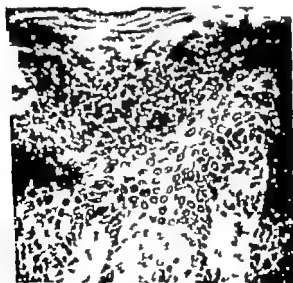


FIG. 57

Psoriasis—The stromal granuloma is absent and replaced by fairly dense collection of polymorphs, the cells of the rete are intact. There is perivascular lymphocytic infiltration in papilla.

Henshaw and Egan $\times 220$.

PITYRIASIS ROSEA.

(Figs 58 and 59)

While the clinical appearances of the lesions in this disease are distinctive, the associated histological changes are slight and ill-defined. The process is that of a mild inflammatory lymphocytic reaction in the papillary portion of the dermis, with an associated slight inter- and intra-cellular oedema of the rete, which is also invaded here and there by lymphocytes. In places the oedema proceeds almost to the formation of small vesicles, and the rete presents a spongy appearance. The stratum corneum becomes ribboned and detached from the underlying rete. The reaction in many respects approaches that of a low-grade eczematous reaction of the flexural infective type, and as such can be classed, both from the clinical and histopathological standpoint, as an eczematide.

PITYRIASIS ROSEA.

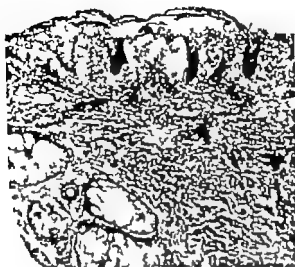


FIG. 58

Pityriasis Rosea.—The stratum corneum is ribboned and detached from the rete, there is some parakeratosis, and the rete is the seat of slight intercellular edema. There is some vascular dilatation and perivascular lymphocytic infiltration in the papillary portion of the dermis. *Henshaw and Essex*. 70

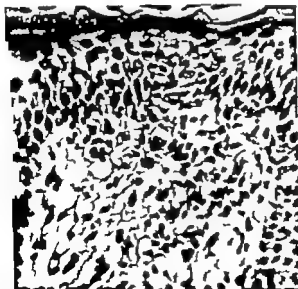


FIG. 59

Pityriasis Rosea.—This shows an area of intercellular edema in the rete, associated with intracellular edema and vacuolation of the cell protoplasm. In some cases rete cell seems to have liquefied completely leaving vacant space, and this area is surrounded by lymphocytes. In the papilla the vessels are dilated and there is an infiltration of lymphocytes. *Henshaw and Essex*. 300

LICHEN RUBER.

The lesion of lichen ruber is a dermo-epidermic papule, which on account of variations in localisation and secondary changes, can be so modified as to produce four distinct varieties of the disease, namely LICHEN RUBER PLANUS (Figs 60-62), the most common type LICHEN RUBER VERRUCOSUS (Fig 63), LICHEN RUBER ACUMINATUS (Fig 64), LICHEN RUBER PEMPHIGOIDES (Figs 65 and 66), an extremely rare modification.

Certain histological features are predominant in all these varieties, and are most clearly seen in *lichen ruber planus*. The stratum corneum is thickened in a regular fashion, and parakeratosis is absent except in lesions of old standing. The stratum granulosum is increased in thickness, sometimes consisting of five to eight layers of granular cells, but the hyperplasia does not occur uniformly being accentuated in some places and little evident in others. The hypergranulosis is evident clinically in the fine network of white lines which can usually be seen on the surface of the papules. The rete is scanty and the pegs are flattened. There is a closely packed infiltration of lymphocytes in the papillary layer of the dermis, and this infiltration is sharply delimited at its deeper and lateral margins. The cells tend to invade the epidermis in the region of the basal layer so that the dermo-epidermic junction is obscured and hazy in places. The tips of the papillae are oedematous, and as a result they become dome shaped, and this, together with the flattening of the rete pegs, causes the dermo-epidermic junction to assume the form of a series of flattened arches. Small collections of fluid may accumulate just underneath the basal layer causing it to split off the dermis, and one of the sections illustrated shows this process accentuated to such a degree as to constitute a bulla, although none was apparent clinically and its presence was not expected in the histological preparation. In the deeper part of the dermis there may be a slight lymphocytic infiltration surrounding the coil glands.

In rare cases the fluid may collect in the rete and raise the thickened stratum corneum so that a clinical and histological intra-epidermic bulla is formed—*lichen ruber pemphigoides*.

In *lichen ruber verrucosus* the same epidermal and dermal changes are present as in *lichen ruber planus*, with, in addition, the upward development of papillary epidermal projections and the formation of horny plugs between them.

In *lichen ruber acuminatus* the lesions have a follicular localisation. Plugging of the mouths of the follicles is conspicuous and the adjacent epidermis is atrophic, probably on account of its being squeezed between

the horny plug above and the infiltration below. Lichen ruber acuminatus may show a clinical resemblance to the mild variety of pityriasis rubra pilaris. The histological difference between the two diseases is quite distinct, for the circumscribed subepidermic lymphocytic infiltration which is characteristic of lichen ruber is absent in pityriasis rubra pilaris.

lichen ruber.



FIG. 60

Lichen Ruber Plaque—There is hyperkeratosis, hyperplasia of the stratum granulosum, which is more prominent in some places than in others. Flattening of the rete pegs, dilated papillae which are edematous, dense circumscribed lymphocytic infiltration in the papillary layer of the dermis, involving the basal layer in places, so that the dermo-epidermic junction is ill defined. Here and there small collections of fluid are to be seen immediately beneath the basal layer, causing clear spaces.
Henshaw and East $\times 60$

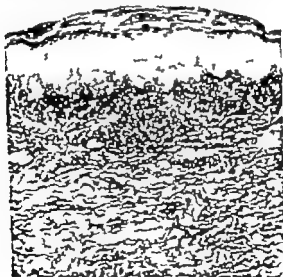


FIG. 61

Lichen Ruber Plaque—Hyperkeratosis and thickening of the stratum granulosum are present. The rete pegs have disappeared, and some of the cells in the deeper strata of the rete are vacuolated. The dermo-epidermic junction is busy. The papillary layer of the dermis packed with lymphocytes and elastic fibres are absent in this area. In the deeper portions of the dermis the collagen and elastic fibres are normal in structure and staining properties. *Lehrhoff and Linn* *Cornell* 80

LICHEN RUBER.



FIG 62

Lichen Ruber Plani.—This section shows large collection of exudate immediately underneath the epidermis. Small collections of fluid are commonly seen in this area. The clinical lesions did not present the appearance of bulla.

Hematoxylin and Eosin $\times 70$.

LICHEN RUBER.



FIG. 65

Lichen Ruber Pseudogomphus.—An intra-epidermic bulla has formed, which is roofed by the stratum corneum. Otherwise the usual features of lichen ruber plaques are to be seen.

Hermann and Euse $\times 12$



FIG. 66

Lichen Ruber Pseudogomphus.—The roof of the bulla is formed by the stratum corneum, and some lymphocytes are present in its cavity. The hyperplastic stratum granulosum seen at the periphery of the bulla, and the sub-epidermic lymphocytic infiltration is seen involving the deeper layers of the epidermis.

Hermann and Euse 90

PITYRIASIS RUBRA PILARIS

(Figs. 67-69)

This appears to be essentially a disease of the epidermis, characterised by a general hypertrophy of all its layers associated with follicular plugging. The condition may be confined to certain areas of the skin surface, or it may become generalised. In the more extensive cases the epidermal change is associated with vaso-dilatation and perivascular infiltration, mainly lymphocytic in type, and the clinical picture is that of a generalised erythrodermia. When an inflammatory phase is in progress on localised areas the disease bears some resemblance to psoriasis, and when in the purely hyperkeratotic and acanthotic types there is a slight tendency to congestion round the orifices of the hair follicles the small lesions may at first sight suggest lichen planus.

Histologically there is a varying degree of acanthosis, associated with marked hyperkeratosis. The grossly thickened stratum corneum dips down into the rete so that the junction between the two zones is represented by a wavy line, and it also distends the mouths of the follicles with horny plugs which may show a whorled arrangement, and which cause some thinning of the rete. The stratum granulosum is uniformly thickened. There may be little or no vascular or cellular reaction in the dermis, and when present it is limited in the mild forms of the disease, to the region of the hair follicle. The cellular infiltration consists mainly of lymphocytes and maintains a perivascular localisation, showing no tendency to collect into masses such as occur in lichen planus.

In the generalised erythrodermatous stage of pityriasis rubra pilaris the epidermal changes are similar to those seen in the milder form, although horny plugs may develop apart from the follicular orifices. The vascular dilation and perivascular infiltration are more uniformly distributed throughout the upper dermis. In both types of the disease the sebaceous glands tend to be atrophic, and the sweat glands scanty.

PITYRIASIS RUBRA PILARIS



FIG. 67



FIG. 68

FIG. 67.—*Pityriasis Rubra Pilaris*.—There is general acanthosis, the stratum papillare is enlarged, and the stratum corneum is many times its normal thickness and dips downwards in places into the rete. There is no vascular or cellular reaction to be seen.
Hematoxylin and Eosin $\times 60$

FIG. 68.—*Pityriasis Rubra Pilaris*.—This section is from a core showing large areas of erythrodermatitis. The usual epidermal features are present; the horny core has developed as reaction to sweat gland orifices. There is diffuse vascular dilatation and perivascular infiltration by lymphocytes in the upper dermis.

Hematoxylin and Eosin $\times 70$.

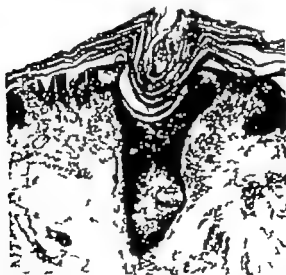


FIG. 69

Pityriasis Rubra Pilaris.—The mouth of hair-follicle is plugged and the stratum corneum forms the plug has lobated arrangement. There is vascular dilatation and perivascular infiltration of lymphocytes along the line of the follicle.

Hematoxylin and Eosin 80

ECZEMA.

Ecze^ma is such a complex subject that it has been thought appropriate not merely to indicate the bare structural changes which it presents, but to correlate them with the clinical manifestations of the disease, and to attempt an analysis of the mechanism by which they have been brought about. A certain part of the explanation offered is applicable also to the *veniculo-bullous* diseases as a whole.

The Ecze^ma reaction is one of the most common examples of cutaneous allergy and the extraneous exciting agent which reacts with the intolerant tissue may reach it from the outside, or from within, *via* the blood stream. Many such agents have been conclusively identified, and many others are suspected with more or less justification, but little is known of the associated biological changes which occur in the tissue cells.

Ecze^ma is an epidermo-dermic reaction which is capable of showing rapid variations in both its clinical appearance and in the histological changes which it presents. To the naked eye the reaction may show three well-defined states, taking the form either of a *vesicular ecze^ma*, a *weeping ecze^ma* or a *scaly ecze^ma*. The scaly phase is common to all eczemas prior to their disappearance, and a weeping ecze^ma may or may not be preceded by one showing clinical *vesiculation*. From the clinical standpoint these three modifications constitute stages of a single and uniform type of inflammatory response. While the reaction itself is limited in its phases it presents additional individual characteristics according to the exciting agent. This factor determines the site on which the reaction develops or for which it shows a preference, its mode of onset and spread, the contours which it assumes, its duration, and the surface area which it affects, so that it is possible to subdivide the majority of cases of ecze^ma into three well-defined groups. These are as follows —

- (1) Chemical ecze^ma
- (2) infective ecze^ma, which can be further subdivided into *fleural infective ecze^ma*, *post traumatic infective ecze^ma* and *follicular infective ecze^ma*, the two latter groups being characterised by *nummular patches* on non-fleural areas
- (3) *diffuse papular ecze^ma*, including *Biemer's Prurigo*.

To avoid confusion due to a possible misunderstanding of the clinical classification of the disease which has been adopted, and which is to be correlated with the histopathological illustrations, a brief clinical resumé will perhaps be advisable.

By chemical ecze^ma is meant an ecze^ma reaction evoked by external contact with a chemical substance to which the skin is allergic or intolerant. In these circumstances the reaction assumes its most typically *vesicular* form, the *vesicle roofs* are intact, and as a result there

may be little initial redness, even though the capillaries are markedly dilated. The presence of a flare forming a halo to the main centrally placed vesicular reaction indicates an accompanying arteriolar dilation. The site of chemical eczema depends on the site of contact of the chemical.

Flexural infective eczema occurs in the body folds and creases. It develops slowly and presents a clear-cut well-defined edge, without a surrounding arteriolar flare. Formed vesicles are scarcely to be seen, and then only in the earliest stages. Their place is taken by pinhead-sized pits in the surface, which ooze copiously and intermittently in the acute phase.

Both varieties of nummular infective eczema are most commonly seen on the backs of the hands and forearms and consist of one or more round patches, which have a clear cut margin, and no surrounding arteriolar flare. As crusting is a prominent feature, formed vesicles are not often seen, and vesiculation proceeds underneath the crusts. The vesicle roofs are poorly formed and easily rubbed off revealing superficial pits. The lesions in this type of eczema develop at localised points and enlarge slowly at their periphery.

While the preceding varieties are regarded as being due to excitants acting on the skin from the outside, in generalised papulo-vesicular eczema an internal origin is more probable. The eruption shows a symmetry of distribution and a constancy in the sites it involves (e.g.—Infantile eczema, Besnier's prurigo). In type it consists of numerous papulo-vesicular nummular patches or isolated small groups of papulo-vesicles. These are well-defined in outline, and a marginal flare is seldom present. The course of the eruption is prolonged and characterised by exacerbation and remission.

The most striking structural alterations associated with eczema are to be seen in the epidermis, where the repeated formation of multiple vesicles, evidence of their attempted formation, or the presence of changes which are related to their formation or previous existence, constitute the basic and predominant histological features of the reaction.

Newly formed vesicles give the appearance of clear spaces in the epidermis which vary in size according to their stage of development, and according to whether coalescence between neighbouring vesicles has taken place. The interior of the vesicle is clear except for a few lymphocytes, an occasional leucocyte, and degenerated epidermal cells—which are swollen, stain poorly, show pyknotic nuclei or are anuclear and which occur singly or in small clumps (Fig. 70). These vesicles may occupy almost the entire substance of the rete mucosum, or be situated about its middle or superficial third, and they occur both in the suprapapillary portion and in the interpapillary pegs. The cells forming their walls are usually flattened but may be swollen and present a degenerated appearance (Fig. 71). The rete in the neighbourhood and underneath the vesicle may be firm and compact (Fig. 73), or it may show intercellular oedema,

with the intercellular spaces much widened and the prickle cells stretched apart (Fig. 77). In some cases the vesicle is separated from the dermis only by a single layer of raggedly arranged and degenerating epidermal cells. In addition to intercellular oedema there may be areas where the cells are hydropic and appear to be saturated with fluid, and in which the nuclei show pyknosis and fragmentation, or have disappeared (Fig. 71). The roof of the vesicle may consist of an intact stratum corneum (Fig. 70), or the vesicle contents may have burst into it, separating its layers (Fig. 72). On the other hand the roof may be composed of parakeratotic cells, a formed stratum corneum being absent (Figs. 81 and 82). In the latter case the vesicle contents easily reach the surface where they coagulate and desiccate, and form into a large crust which covers a number of vesicles (Fig. 75). The crust may show alternating areas of coagulated tissue-fluid and parakeratotic cells, the whole overlying a vesicular rete—a picture which indicates the repeated and successive formation of vesicles and the continuing nature of the reaction (Fig. 78).

The epidermis overlying the papillae may be represented merely by a few widely separated, degenerated cells, so that there appears to be a direct communicating channel between the oedematous and congested papilla and the exterior (Figs. 83 and 88).

The whole epidermis may be acanthotic, and when this occurs numerous cells in the area show active mitosis (Fig. 80).

In the papillary portion of the dermis the capillaries are dilated, the papillae may be oedematous, and the degree of oedema may sometimes be extreme (Figs. 81, 82 and 84). There is a variable amount of perivascular lymphocytic infiltration. On the whole, however the dermal changes may be extremely slight, even when the clinical intensity of the reaction is considerable and the most striking changes and extreme structural alterations are to be found in the epidermis (Fig. 70).

The rapidity with which these structural changes may develop is illustrated by Fig. 70, which shows fully developed vaciculation within twelve hours of contact with the exciting cause, and in Fig. 86 which shows a reconstitution of the cellular disturbance depicted in Fig. 85 within a period of thirty-six hours. In urticaria even more rapid changes from normal to pathological and vice versa are to be seen clinically but in urticaria gross structural alterations and evidence of cellular damage are absent (Fig. 43 Urticaria), and the lesion depends entirely on a vasomotor reaction involving transient but gross variations in capillary filtration.

The foregoing is a general review of the disorganisation of the cutaneous structure which constitutes the eczema reaction, but just as there are clinical modifications which allow the disease to be divided into three main groups, each of which seems to depend on the nature of the exciting agent, so it is found that the histological picture is correspondingly modified in each of the groups, some of its features being accentuated, others being partially in abeyance.

These particular modifications in the general character of the eczema reaction can readily be appreciated from the illustrations, which have been arranged according to the type of case from which the tissue was obtained. Thus in chemical eczema vesiculation takes place under an intact stratum corneum and is seen in its most complete and highly developed state.

In nummular infective eczema vesiculation occurs superficially in the epidermis and the vesicle contents escape early on to the surface to dry up and form a crust, under which the process continues. Gross surface infection of a secondary nature may be responsible for the development of a dense leucocytic infiltration (Fig. 79), but, although eczema of this type is presumed to be in part at least of bacterial origin, a leucocytic reaction is a rare accompaniment.

In flexural infective eczema oedema of the papillary portion of the dermis may be extreme (Fig. 84) and cellular oedema of the epidermis is much in evidence (Fig. 85). The vesicles are small, situated superficially in the epidermis, and roofed by degenerated cells, or by dried serum (Figs. 81 and 82). Considerable disintegration and atrophy of the whole epidermis may be apparent in long standing cases (Fig. 87). The most distinctive feature in this type of eczema is the loss of epidermal cells at the tips of the papillae, with the formation of channels leading from these structures to the surface, which are evidenced clinically as "pits" (Figs. 83 and 88).

The fact that both nummular infective eczema and flexural infective eczema present an intense redness from their commencement is due not only to vaso-dilatation, but to the early loss of the capacity to form a stratum corneum.

In the diffuse papulo-vesicular type the structural changes vary between those of a nummular and a flexural eczema.

It may be objected that these three characteristic modifications of a general histological picture merely indicate its consecutive or inter-related phases and there is no doubt that each modification may predominate at some stage in the same patch of eczema. Nevertheless the clinical groups with which they are constantly associated present such individuality in their rate of development, course, response to treatment, and as far as can be ascertained as regards their cause, that any structural modification which is in a general way peculiar to any one of them should meantime be regarded as significant and as having some relationship to the biological processes by which the reaction has been brought about. Furthermore they may be interpreted as bearing the imprint of the general character of the causative agent. That they do not depend entirely on the peculiarities and susceptibilities of an individual skin is demonstrated by Figs. 73, 80 and 88, which show the three modifications due to different exciting factors, occurring almost simultaneously in the same patient on separate sites. On the other hand a transformation from one type of reaction to another may occur in the same eczema lesion: thus a nummular eczema may assume the clinical and histolo-

gical features of a flexural type of reaction. It is difficult for a typical chemical type of reaction to develop on a pre-existing nummular or flexural type, because of the already existing epidermal changes and the absence of a stratum corneum. What happens in these circumstances is an exaggeration of the existing reaction, with a tendency for it to assume the flexural type with plus, and the development of a typical chemical reaction in the immediately surrounding intact skin.

THE FACTORS INVOLVED IN THE PROCESS OF VESICULATION

The mechanism by which the recurrent vesiculation of eczema is brought about can be deduced to some small extent from a study of the histopathology of the reaction in its various forms and stages, from a consideration of the associated clinical data, and from a comparison with these factors as they obtain in other superficial exudative inflammatory diseases. If the size of the vesicle in Fig. 70 is compared with the adjacent rete it is obvious that the area now occupied by the vesicle must have been represented originally by a large number of cells. It is difficult to conceive that an influx of capillary filtrate from the dermal capillaries could have overcome existing intra-epidermal tissue pressure and exerted sufficient hydrostatic pressure to disrupt the normal intercellular cohesion of the rete cells with the formation of a fluid-containing space of such dimensions, in the absence of other factors. There is no constant or gross intercellular oedema in the cellular layer forming the floor of the vesicle to suggest a passage of filtrate—actually an examination of the eczema vesicles illustrated, and of Fig. 122, which shows the base of an impetigo bulla, reveals the fact that intercellular oedema may be entirely absent in this area. The cells forming the walls of the vesicles are flattened and closely approximated, and the layer of flattened cells is not extensive. These facts suggest that the cavity is in part the result of liquefaction of rete cells, and to support this hypothesis there is visual evidence of localised groups of rete cells well advanced in degeneration (Fig. 74). That there is localised increase in tissue fluid, and a disturbance in its distribution is shown by the localised areas of intercellular oedema (Fig. 77) and by evidence of oedema in the papillary portion of the dermis (Figs. 81, 82, 83, 84, 85 and 87).

An increase in capillary filtrate is brought about by an increase in the permeability of the capillary wall, in the hydrostatic pressure within the capillaries, and in the rate of blood flow through them, which in turn is dependent on the state of dilatation of the arterioles. In all forms of eczema the capillaries are dilated. In chemical eczema a flare is seen at the periphery of the lesion, indicating an accompanying arteriolar dilation, but in nummular and flexural eczema there is no such flare to be seen at the edge of the lesions so that in these types no arteriolar dilation of any significance can exist, and an increase in capillary pressure and rate of blood flow from this cause cannot take place. A similar absence of flare is seen in the fresh bullae of pemphigus. Capillary

dilatation without a simultaneous arteriolar dilatation or venous occlusion can only result in a fall in capillary pressure so that hydrostatic pressure can play little active part in the localised accumulation of fluid, and from the vascular side an increase in the permeability of the capillary walls can be the sole operative factor in bringing this about.

An increase in hydrostatic pressure, in the rate of blood flow and in capillary permeability all occur in ordinary urticaria, and the result is a localised transient oedema of the papillary portion of the dermis. (Fig 44).*

If in urticaria, where the most favourable conditions exist for the development of increased capillary filtration, the filtrate is limited to the dermis, where it so increases tissue pressure that the hydrostatic intracapillary pressure is ultimately overcome and the capillaries collapse, and yet the epidermis is not normally penetrated, it is obvious that changes in hydrostatic pressure relations in the eczema reaction cannot alone account for the presence of fluid in the epidermis. In eczema the dermal tissue pressure does not rise sufficiently to overcome capillary pressure. The factor which attracts fluid into the epidermis must therefore be some physico-chemical force which has developed as a result of the localised necrosis and liquefaction of epidermal cells, a state of affairs which is revealed by histopathological study

In eczema the variable degree of epidermal and dermal oedema seen in different specimens in association with equally well developed vesicles, the histological evidence that vasculature is a continuously repeated process *in situ* and the known rapidity with which structural changes can occur suggest that the flow of tissue fluid into the epidermis occurs intermittently and that the process is somewhat as follows—Multiple localised foci of epidermal necrosis are produced in association with or as a result of the allergic process. A physico-chemical force develops in these areas and sucks the adjacent tissue fluid to them, capillary permeability is increased and the filtrate is also pulled to the necrotic areas. The passage of the capillary filtrate through the epidermis to localised points is evidenced by transient intercellular oedema. The commencing localisation of this fluid, together with that derived from liquefied epidermal cells in a commencing vesicle is also responsible for the temporary appearance of intercellular oedema, which is soon replaced by a pool of fluid as the vesicle matures. The increasing hydrostatic pressure within the developing vesicle gradually overcomes the attracting force, and the movement of fluid within the epidermis stops for the time being, the size of the vesicle being limited by the elasticity of its walls and by the surrounding tissue pressure. The entire process is repeated by the development of fresh areas of epidermal necrosis again upsetting the equilibrium of the tissue fluid and capillary filtrate.

*Eczema urticaria of such rare occurrence and obviously involves factors which are absent in ordinary urticaria that requires special consideration and can be excluded from the present comparison

A comparable periodicity of an allergic process is known to occur in urticaria where there is complete clinical disappearance of the lesions between the attacks. In urticaria a rapid reabsorption of the capillary filtrate in the dermis is possible, due to the influence of the colloid pressure of the blood plasma, for there is no histological evidence of damage to cellular structures such as would add colloid to the exuded filtrate and so hinder its reabsorption. The lymph vessels offer a further channel for absorption, and the filtrate surrounds these vessels and the capillaries, or is in their near vicinity. In eczema the fluid eventually concentrates at a distance from the vascular and lymphatic systems, is separated from them by compact cellular tissue, its colloid content is considerable, therefore reabsorption does not take place.

In urticaria there is a periodic transient diffuse soaking of the upper dermis with capillary filtrate, associated with the temporary development of increased tissue pressure. In eczema there is probably a similar periodic transient soaking of the epidermis with capillary filtrate, which is at first diffuse but which is rapidly concentrated in localised areas to form, along with the liquified epidermal cells, the vesicles which are typical of the reaction. The type of modification of the reaction which is associated with the individual clinical groups must depend on the nature and site of the initial cell damage which in turn must be governed by the exciting factor and the dominant features of the allergic process. The fact that reabsorption of the oedema in eczema is difficult, with the result that restitution of the damaged tissue to normal is slow may tend to render the vascular phenomena associated with it more continuous than in urticaria.

This conception of a periodicity in the fluid movement in eczema comparable to that which from clinical observation, is known to occur in urticaria, would account for the varying pictures of vesiculation with and without an accompanying intercellular and papillary oedema, the presence or absence of which would depend on the state of activity of the eczema process at the time of removal of the tissue for examination.

The ultimate fate of the unabsorbable fluid collections in the epidermis is either extrusion to the exterior by rupture of the vesicle roof or desiccation *in situ*, the determining factor being the site of the vesicle in the epidermis and the integrity of the tissue forming its roof. The state of the cellular layer forming the roof of the vesicle depends in turn on whether the reaction takes place in a hitherto intact epidermis, or in one which has been subject to recurrent oedema. In the latter case the tissue in which the factors necessary for vesicle formation develop may be in such a state of disintegration that only imperfect or abortive vesiculation is possible (Fig. 87), and indeed there may be direct communicating channels conducting the capillary filtrate from the papillae to the exterior. It has already been noted that this latter development is peculiar to flexural infective eczema, in which the

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transient nature and periodicity of the increase in capillary filtrate can be observed clinically in the sudden alternation of dry and wet phases.

Similar factors may govern not only the final disposition of the eczema vesicle, but also that of the vesicles and bullae of dermatitis herpetiformis, herpes zoster herpes simplex pemphigus, impetigo and varicella.

In eczema as soon as the oedema subsides the epidermal cells rapidly become reconstituted as a compact mass, and as a result of mitosis the entire rete becomes thickened (acanthotic) (Fig. 86), but it may be some considerable time before the normal mode of evolution of the cells from the basal layer to the surface is re-established and a normal stratum corneum produced. During this stage of retrogression the surface is composed of a parakeratotic layer and the eczema is clinically in a scaly stage. If the eczema reaction repeatedly attacks the same area, the latter may become hypertrophied (Fig. 91 lichenification), but as this is by no means a constant sequel it would seem that some further cutaneous peculiarity is necessary for its occurrence. It is seen in its highest degree of development in the chronic constitutional eczema known as Besnier's prurigo, a condition which not infrequently occurs in association with ichthyosis, and one which is often accompanied by asthma.

ECZEMA—CHEMICAL



FIG. 70

Chemical Eczema—Vasodilation is fully developed within twelve hours of contact with the irritant (mercury), and involves almost the entire thickness of the epidermis. The stratum corneum is intact. The cells in the immediate vicinity of the vessels are flattened. There is no obvious dermal edema, the vessels are dilated and there is moderate perivascular accumulation of lymphocytes. *Hematoxylin and Eosin* $\times 100$



FIG. 71

Chemical Eczema—A high power view of the septum between the large vessels in Fig. 70 and the smaller one situated on its right side. The cells are flattened, the cytoplasm is indistinct and stains poorly; the nuclei are pyknotic, an area of necrosis and liquefaction with loss of cell outline and nuclei is to be seen in the bottom right hand portion. The vessel contains lymphocytes, degenerated epidermal cells (top left), and cell debris. *Hematoxylin and Eosin* $275\times$

ECZEMA—CHEMICAL



FIG 72

Chemical Eczema—Eczematous reaction to mercury twenty-four hours after contact showing well formed vessels rupturing into the layers of stratum corneum. With the exception of some intercellular edema and decrease of individual epidermal cells at the base of the vessels, the surrounding cells are reversibly intact.

Henshaw and Essex $\times 275$



FIG 73

Chemical Eczema—Showing the same structural changes as in Figs. 71 and 72.

Henshaw and Essex 100

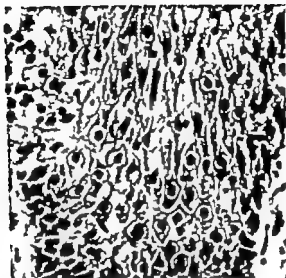


FIG. 74

Chemical Eczema, cornifying nuclei—The skin cells are edematous, vacuolated—much as evidence, the nuclei are pyknotic and in some cells they have disappeared. few nucleolar cells are in process of disintegration.

Hamman and Ems 420



FIG. 75

Nummular Infective Eczema.—A small patch of nummular infective eczema of thirty-six hours duration. A large mass of dried exudate rests on an acanthotic epidermis in which are numerous small vesicles and areas of intercellular edema. The dermis shows dilated vessels and slight perivascular lymphocytic infiltration.
Hematoxylin and Eosin $\times 75$.

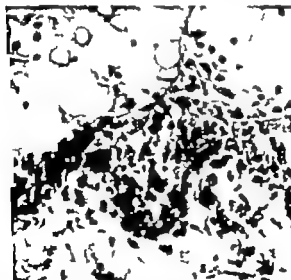


FIG. 76

Nummular Infective Eczema.—High power view of the epidermis underlying the crust in Fig. 75. It shows intercellular edema, multiple small vesicles containing degenerating epidermal cells, and lymphocytes within the vesicles and penetrating the epidermis.
Hematoxylin and Eosin 350.

ECZEMA—INFECTIVE.

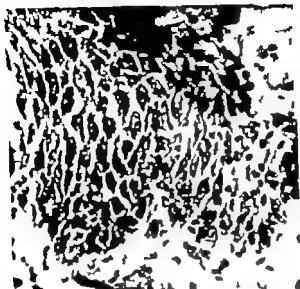


FIG 77

Nonspecific Infective Eczema—A high power view of the epidermis underlying the crust in Fig 75. It shows intercellular edema, intracellular edema, degeneration and disappearance of individual epidermal cells, and invasion by small number of lymphocytes. *Hematoxylin and Eosin* $\times 550$



FIG 78

Nonspecific Infective Eczema—Nonspecific patch of eczema of six weeks' duration. There crust of dried exudate on the surface resting on a layer of parablastic surface cells. A vesicle present superficially on the epidermis and contains degenerated epidermal cells and few lymphocytes. The contents of the vesicle are rupturing into the parablastic surface. The epidermis forming the base of the vesicle shows intercellular edema, degeneration of the epidermal cells, and infiltration by lymphocytes. The hole picture indicates recurrent vesicle formation. *Hematoxylin and Eosin* 275



FIG. 79

Numbular Infective Eczema.—Numbular patch of similar acute duration. The alteration in structure is similar to that shown in Fig. 75 but there is a gross leucocytic reaction in the superficial dermis, indicating secondary infection with organisms which have no connection with the production of the eczematous reaction.

Hamman and Egan $\times 100$



FIG. 80

Numbular Infective Eczema.—Showing the same structure as Fig. 75 and in addition considerable acanthosis.

Hamman and Egan $\times 90$

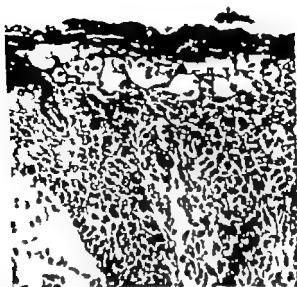


FIG. 81

Flaccid Infective Eczema of ankle of one month duration—This shows multiple small vesicles situated very superficially in the epidermis and roofed by a thin layer of sodden flattened nucleated cells. There are degenerated epidermal cells in several vesicles. Intercollicular edema is visible on one side of the papilla, but there is compact rete on the other side. A dilated capillary appears in the papilla surrounded by lymphocytes which are also invading the rete. *Hermann and Egan* 225



FIG. 82

Flaccid Infective Eczema—An adjacent field to that shown in Fig. 81 showing similar vasculature, acanthosis, and areas of rete cells undergoing degeneration and soaked with exudate. There is considerable edema in the papillary layer of the dermis. *Hermann and Egan* 120

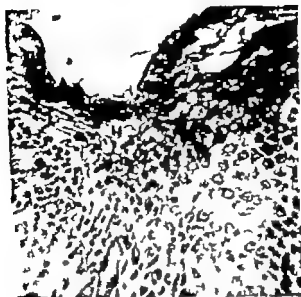


FIG. 83

Flexural Infective Eczema.—An adjacent field is shown above in Figs 81 and 82. Here there is a channel leading from the surface to the tip of papilla—an abscess pit. The adjacent area to the right shows superficial vasculature, the underlying rete being normal in structure. *Hawthorn and Eames*. 300



FIG. 84

Flexural Infective Eczema.—From the same case of acute weeping eczema of the axilla as in Fig 81 to Fig 83. There is no vasculature to be seen in this field, but the more superficial cells in the rete stand poorly. Coarse papillary ridges in the striking feature. *Hawthorn and Eames*. 300



FIG. 85

Flexural Infective Eczema.—From case of acute weeping eczema of the retro-auricular region, of four days duration. There is marked intercellular edema and cell degeneration, the stratum corneum is replaced by a layer of flattened nucleated cells and the papillae are edematous. *Hammann and Essex* 275

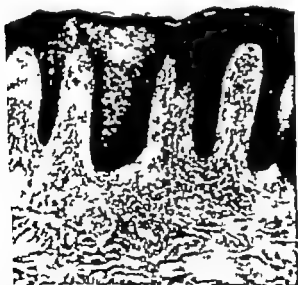


FIG. 86

Flexural Infective Eczema.—The same case as Fig. 85, thirty-two hours after treatment with wet dressings of 0.5% solution of silver nitrate in distilled water. Epidermal and papillary edema have subsided and there are no areas of cell degeneration in the epidermis. The rete forms compact mass, has become anasthetic, and stratum corneum attempting to form. *Hammann and Essex* 300

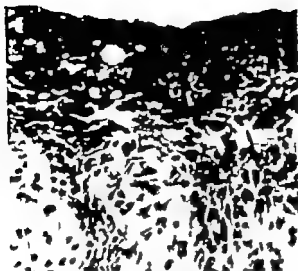


FIG. 87

Flaccid Infective Eczema.—From case, primarily affecting the retro-auricular regions and groin, which became generalized. The duration of the disease was two years, and recovery was satisfactory. The specimen illustrated was taken from the abdomen in the region of the groin when the eruption was in an acute exsiccative phase one year from the establishment of the eruption over the entire body. The epidermis is fragmented, there is cellular degeneration, and small imperfectly formed vesicles are present in the rete. There is direct communication between the overlying mass of dried exudate and the top of the central orificious papilla. Lymphocytes are scattered throughout the lesion, but polymorphs are practically absent.

Hematoxylin and Eosin $\times 300$



FIG. 88

Flaccid Infective Eczema.—Showing similar changes to those in Figs. 87 and 83. The radiate or be dermal papilla well shown and at one point there is break through the epidermis and continuity with the exudate on the surface.

Hematoxylin and Eosin 100

ECZEMA—GENERALISED EXFOLIATIVE.

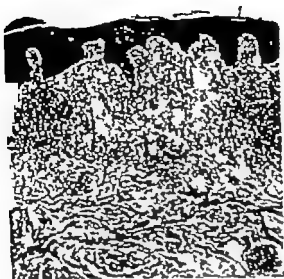


FIG. 89

Generalised Exfoliative Eczema—One year duration. The epidermis shows irregular acanthosis with broadening of the rete pegs and parakeratosis. There is evidence of the papillary layer of the dermis, together with vascular dilatation and moderate and moderately dense infiltration by lymphocytes. The papillae are dome shaped and few lymphocytes are crowding the epidermis. Compare with Psoriasis, and generalisation of flexural ecthyma eczema (Figs. 55 & 81-86).

Hematoxylin and Eosin $\times 75$.

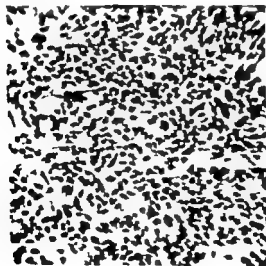


FIG. 90

Generalised Exfoliative Eczema—The infiltration of the dermis almost entirely lymphocytic.

Hematoxylin and Eosin 330

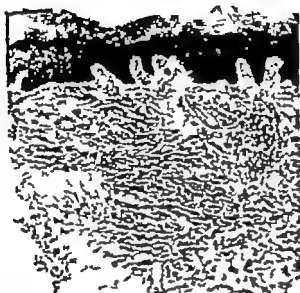


FIG. 91

Besnier Prurigo—The lichenified tissue is from the elbow fold in case of *Besnier Prurigo* during remission. There is marked hyperkeratosis, some degree of acanthosis, and slight perivascular lymphocytic infiltrate.

Hematoxylin and Eosin $\times 75$

CHEIROPOMPHOI YX.

This term, and also that of Dyshydrosis, is used to indicate a vesiculo-bullous eruption affecting the palms of the hands, the sides of the fingers, and the soles of the feet. The eruption is of sudden onset, and in some cases has a tendency to recurrence. It was originally regarded as an affection of the sweat glands, but this view is not supported either by clinical observation or histopathological study. The microscopic appearances are those of the eczema reaction as it is evoked by chemical irritation, the fact that the process of vesiculation takes place in an epidermis with a specially well-developed stratum corneum ensuring the formation of structurally intact vesicles (Fig. 92). From the etiological standpoint the condition is in the majority of cases attributable either to chemical irritation, to a fungus infection, or to the irritant action of noxious substances reaching the skin from within, e.g., toxins absorbed from areas of fungus infection in other parts of the skin surface, or from areas of focal sepsis of bacterial origin.



FIG. 92

Cheirorhynchus.—The structural changes are those of the
 reverse reaction as it is evaluated by chemical analysis
Harrison and Egan $\times 65$

PRIMARY VESICULO-BULLOUS ERUPTIONS OF INTERNAL TOXIC ORIGIN

This group of diseases includes Herpes Zoster Varicella, and Variola Erythema Multiforme, Dermatitis Herpetiformis, Pemphigus Vulgaris, and the Senear Usher syndrome and Keratoderma Blennorrhagica. The eruptions which depend on cutaneous idiosyncrasy to ingested drugs, particularly iodides, may occasionally present a primary bullous character but the effect of these drugs is erratic, and the eruptions which they produce do not constitute specific clinical entities. In addition, a variety of cutaneous disorders of internal origin, which are not primarily bullous, may from time to time develop bullae as a secondary phenomenon—e.g., urticaria and lichen planus.

In those diseases which are vesiculo-bullous from their onset it seems that the toxin or virus which produces them has a destructive action on the cells of the rete, causing them to become hydropic and vacuolated, and bringing about degenerative changes in the nuclei. These changes may even proceed to a stage of coagulative necrosis. The lesions which result from this action are characteristic for the various diseases as regards their clinical appearance and course, but microscopically the distinction is less clear cut, and they are to be differentiated more by the degree or stage of the degenerative process than by any specific or outstanding difference in the general effect of the causative agent on the cells of the rete, or in the type of vascular and cellular response which it may elicit in the dermis.

HERPES ZOSTER (Figs. 93-95) Preceding the development of intra-epidermic vesicles and bullae the rete cells undergo a series of degenerative processes. These consist in vacuolation, pyknosis and karyorrhexis, swelling of the cells, altered staining properties, and coalescence of degenerated cells with the formation of spaces or large multi-nucleated cells. Eosinophilic droplets may appear in the nuclei and in the cell cytoplasm. The degenerative process which affects the cells forming the base of the bulla may be so complete that its floor is ultimately formed by the bare dermis. In the bulla are to be found ballooned, multi-nucleated, and degenerated rete cells, floating free in the fluid contents. There is an intense leucocytic cellular reaction under the bulla, the inflammatory cells invade the rete, and the contents of the bulla may become purulent. There is vascular dilation and oedema in the dermis, where there may also be haemorrhage and necrosis. The lesion heals with the formation of a scar which may be depressed below the surrounding skin surface if necrosis, apart from epidermal degeneration, has been a marked feature.

VARICELLA (Figs 96-99). The lesions in this disease are identical with those of herpes zoster. The vesicles and bullae are more frequently confined to an intra-epidermal situation. Swollen and multinucleated cells are present in the base of the blister and in its contained fluid vacuolation is of frequent occurrence, and hyaline eosinophilic intra-nuclear bodies may be seen.

VARIOLA AND VACCINIA (Figs. 100-103) In these closely allied lesions the degenerative epidermal changes are more intense than in herpes zoster and actual necrosis with complete epidermal destruction is a feature. The swollen and multinucleated rete cells which are common in herpes zoster and varicella are much more rarely seen in variola, and in the latter disease extra-nuclear eosinophilic droplets (Guarnieri bodies) are to be found *in the cell cytoplasm* in contrast to similar structures situated *within the nuclei* in herpes zoster and varicella.

ERYTHEMA MULTIFORME (Figs. 104-106) In this disease the histological features are mainly centred in the epidermis. Over a circumscribed area, corresponding to the size and shape of the clinical erythematous-bullous plaques, the epidermis is honeycombed with fluid containing spaces of varying dimensions, the whole rete being thus disorganised by a series of multilocular bullae. The walls of these lacunae are formed by oedematous and partially degenerated rete cells, and the partitions between them may consist of little more than a thin strand of epidermis. The stratum corneum is intact. In addition to this inter- and intra-cellular oedema the entire affected epidermis with the exception of its rete pegs, may be raised from the dermis by an exudate of plasma in which may be found strands of degenerated rete-cells, a few lymphocytes and leucocytes. Associated with this severe epidermal upset there is a remarkably slight inflammatory reaction in the papillary layer of the dermis, with a scanty perivascular infiltration of lymphocytes, and slight vaso-dilatation. It seems as if some unknown agent has exerted a profound toxic action on the rete, and that the vascular response to this has been almost exclusively one of increased capillary filtration the oedematous fluid being localised throughout the deepest layers of the rete with the production of a bulla, the roof of which is formed by almost the entire thickness of a disorganised rete. The epidermal necrosis in this disease has features in common with that seen in variola, and varicella.

DERMATITIS HERPETIFORMIS (Fig. 107). The lesions in this eruption may be purely erythematous, urticarial, bullous, or a mixture of all three types. Scarring is a common sequel in the bullous lesions due to the fact that they are frequently in a sub-epidermic situation. The cellular reaction in the dermis is lymphocytic in type, but there are a varying number of polymorphs present in the tissue forming the base of the bullae. The blood shows a moderate eosinophilia, and eosinophils may be present in the bulla fluid. In addition to scarring the lesions

may cause hyperpigmentation, and melanophores may be seen in numbers in the dermis.

PEMPHIGUS VULGARIS (Figs. 108 and 109). The bullae are situated in the middle and deep zones of the epidermis, and in some cases they are practically sub-epidermal. They are generally unilocular and isolated rete cells in their walls and base may be vacuolated and ballooned, and some may have coalesced to form multi-nucleated cells. Even when Nikolski's sign is present (friability of the apparently intact epidermis in the vicinity of bullae, and the ability to make the bullae travel along the epidermis by pressing on their sides), there is no visible lack of cohesion in the surrounding rete beyond perhaps a slight degree of intercellular oedema in the immediate vicinity of the bulla. A moderate cellular infiltration is present in the oedematous papillary layer of the dermis underneath the bulla leucocytes predominate, while in the perivascular areas the infiltration is composed of lymphocytes. In the vegetating variety of pemphigus vulgaris (pemphigus vegetans) verrucous excrescences develop from the exposed bases of the ruptured bullae, and the cellular infiltration in the dermis is more intense. In pemphigus the blood shows a marked eosinophilia.

SENNER USTHER SYNDROME (Figs. 110 and 111). This condition is a rare complication of lupus erythematosus and in its clinical manifestations it bears some resemblance to pemphigus. It is also referred to as pemphigus foliaceus. The bullae are so superficially placed in the epidermis that the roof is soon ruptured and remains as a flaccid epidermal tag. The shape of the lesion is usually that of a crescent. Histologically the bullae are seen to develop immediately below the stratum corneum and after rupture has taken place the exposed surface of the rete is ragged and eroded. The epidermis is acanthotic and here and there the rete cells are oedematous, otherwise its structure is normal. There is a slight inflammatory reaction in the papillary layer of the dermis.

KERATODERMIA BLENNOGONORRHOICA (Figs. 112-116). This eruption occurs as a complication of gonorrhoea, is generally situated on the soles and palms, and is occasionally sparsely distributed on the legs, trunk and arms. The individual lesions last from three to ten weeks or longer. They commence as vesicles which become purulent and are transformed into an adherent crust. Their slow development and regression can be traced histologically and the most striking features are a marked degree of acanthosis with elongation of the rete pegs, and an infiltration of polymorphs, histiocytes, and lymphocytes which traverse the rete to become incorporated in layers and clumps in the overlying crust. The crust is composed of fibrin and dried plasma, inflammatory cells and parakeratotic epidermal cells. In the earlier stages vesiculation continues to take place beneath this crust, which thus becomes increasingly thickened. The papillary layer of the dermis is grossly oedematous.

HERPES ZOSTER

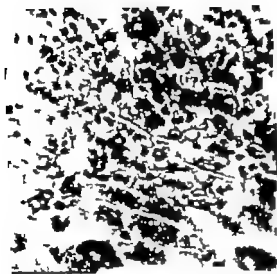


FIG. 93

Herpes Zoster—Floating free in the body fluid are lymphocytes, swollen red cells in various stages of degeneration, red-streaked red cells, and fragments of nuclei.
Hammon and Evans 340

VARICELLA



FIG 96

Varicella—*Early papulo-vesicular stage*. There are several large multilocular vesicles situated immediately under the stratum corneum. These collections of fluid rest on the entire thickness of the rete, the cells of which are undergoing disintegration and necrosis. The subjacent dermis is edematous in its papillary portions, and in its deeper layers there is moderate perivascular lymphocytic infiltration.

Hammon and Essex 65



FIG 97

Varicella—*Early papulo-vesicular stage*. Oedema fluid has collected beneath the stratum corneum to form large vesicles. The cells of the underlying rete are disintegrated in many places. They stain poorly; the intercellular bridges are no longer present, and in many instances the cell membranes have disappeared. The nuclear chromatin is lost, and the nuclei are represented by their membranes and nucleolus. In places the cell nuclei and nuclei have disappeared entirely and the space is filled by mass of poorly staining cytoplasm.

Hammon and Essex 225

VARICELLA.



FIG. 98

Varicella—Pustular stage. Except at the edge of the lesion the entire rete is replaced by a pool of fluid, presumably composed of exudate fluid and liquefied rete cells. The roof of this bubble is formed by the stratum corneum and in the deeper areas there is mass of nuclear debris, lymphocytes, and an occasional polymorph and histocyte. The epidermal process is sharply demarcated from the adjacent rete. The papillary layer of the dermis at the edge of the necrotic area the seat of lymphocytic infiltration.

Henshaw and Essex 65

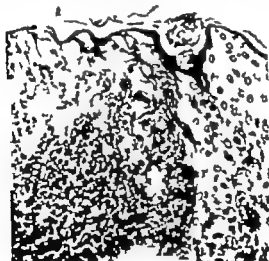


FIG. 99

Varicella—Pustular stage.—T the right of the section the rete cells and their nuclear stain poorly and the cell outlines are ill-defined. Here and there cells which have undergone an eosinophilic degeneration are to be seen surrounded by clear spaces. T the left, the rete cells have disappeared, their place being taken on the superficial area by homogeneous eosinophilic fluid roofed by the stratum corneum and in the deeper area the fluid contains large amount of nuclear debris and few lymphocytes. Here and there balloon cells can be distinguished in the superficial area of the bulla.

Henshaw and Essex 125



FIG. 100

Vaccin. —The lesion is clearly demarcated from the surrounding skin. The surface consists of an excoriated plasma and beneath this the epidermis is completely necrotic and infiltrated by inflammatory cells.

Henshaw and Egan 7



FIG. 101

Vaccin. Edge of lesion —The adjacent epidermis is acanthotic and the cells are swollen, stain poorly and many are vacuolated. The lesion consists of dried serum in its upper portion, with necrotic epidermis in the deeper areas, the entire mass being infiltrated by inflammatory cells. The papilla are acanthotic and infiltrated by lymphocytes and polymorphs.

Henshaw and Egan 60



FIG. 102

Generalized Vaccinia, pustular stage—The surface of the lesion, composed of masses of vacuolated and completely degenerated rete cells, covered by polymorphs. Several small dried up vesicles are embedded in the mass. The underlying rete is anastomotic, and contains vacuolated cells, and cells which have degenerated to form homogeneous mass. The papillae are anastomotic, and both these and the adjacent dermis contain dense infiltrate of polymorphs and mononuclears. *Hematoxylin and Eosin* $\times 75$.

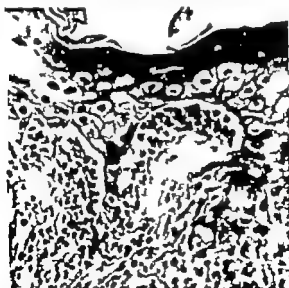


FIG. 103

Generalized Vaccinia, pustular stage—The rete is in state of extreme degeneration. The majority of the cells are vacuolated, some have lost their nuclei, the remaining cytoplasm is granular and stains poorly; the papillae are absent, and the degenerative cells appear to be calcifying. The papillae are anastomotic and packed with inflammatory cells. *Hematoxylin and Eosin* 350.

ERYTHEMA MULTIFORME.



FIG. 104

Erythema Multiforme.—An intense exudative process has developed in association with necrotic changes in the epidermis. The epidermis is honey-combed with fluid accumulations of varying sizes, and, with the exception of the rete pegs, it forms a roof for large bull's heads, the base of which is formed by the papillary layer of the dermis almost throughout its entire extent.

Hamaker and Egan $\times 17$

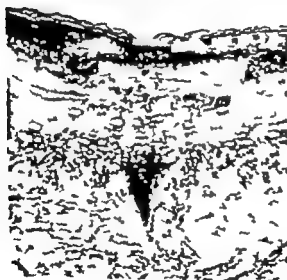


FIG. 105

Erythema Multiforme.—The bull's head is almost entirely sub-epidermal, and the cells in the epidermis forming its roof have undergone degenerative and necrotic changes. The vascular and cellular reaction in the dermis forming the base of the bull's head is exceedingly acute.

Hamaker and Egan 100

ERYTHEMA MULTIFORME.

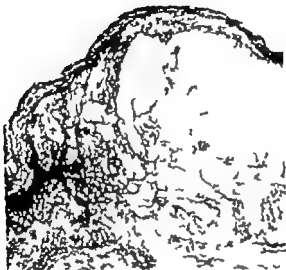


FIG. 106

Erythema Multiforme.—The degenerative changes in the epidermis are sharply defined from the adjacent normal rete. The epidermis is honey-combed with multi-focal bullae, and shows the roof of sub-epidermal bullae. There is slight perivascular infiltration by lymphocytes.
Hammon and Essex 70



FIG. 107

Dermatitis Herpetiformis.—The section shows sub-epidermal bullae. The roof composed of the entire thickness of the epidermis, each has been raised from the dermis by collection of fluid at the dermo-epidermal junction. The base surface of the dermis forms the base, and on it there is a narrow band of lymphocyte infiltration. Deeper in the dermis there is perivascular lymphocytic infiltration.

Harrison and Lown 45

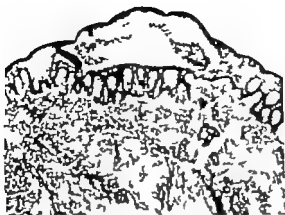


FIG. 108

Pemphigus vulgaris—A bulla has formed in the middle portion of the rete. There is some acanthosis. A moderate histiocytic reaction is present in the underlying dermis. The epidermis surrounding the bulla appears to be normally contacted and there is no apparent lack of cohesion of the cells. Nikolsky sign was not uniformly present in this case.

Hershenov and Egan < 20



FIG. 109

Pemphigus vulgaris—In this example the bulla is almost sub-epidermic in situation. The cellular reaction is minimal. Nikolsky sign could be easily elicited in this case.

Hershenov and Egan 15

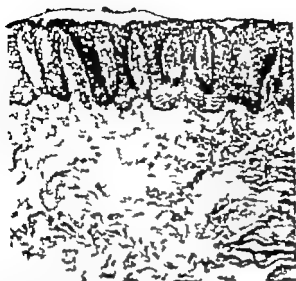


FIG. 110

Sennear Usher Syndrome—The epidermis is atrophied; the rete pegs are absent, but the supra papillary portion of the rete is much reduced in width. The stratum granulosum absent, and the thin stratum corneum is ruled by collection of fluid in furrows. Blood cells. The papillae are absent, and there is slight lymphocytic infiltration. The collagen fibres have homogeneous glassy appearance. *Hematoxylin and Eosin* $\times 75$

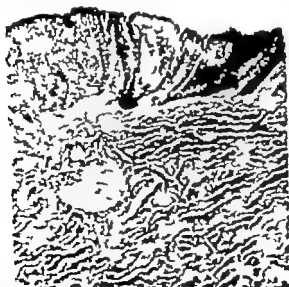


FIG. 111

Sennear Usher Syndrome—The right half of the section shows the base of ruptured bullae; the roof of each has disappeared. The rete and the papillae are both slightly edematous. The stratum granulosum has disappeared, and the exposed surface of the rete is ragged and fragmented. The collagen is somewhat glassy in appearance. *Hematoxylin and Eosin* $\times 75$

KERATODERMIA BLENNORRHAGICA.



FIG. 112

Keratoderma Bleorrhagica, early period, cuticular lesion.—The stratum corneum is intact and forms a covering to mass of consolidated exudate in which are embedded numerous leucocytes and periblastotic cells. The rete cells are edematous, and leucocytes are present in the inter-cellular spaces. There is acanthosis. A leucocytic infiltration is present in the papillary layer of the dermis.

Hannan and Essex 30.



FIG. 113

Keratoderma Bleorrhagica, an early period, vascular lesion.—A recent vessel is seen arising on the rete and is surrounded by older consolidated exudate in which are numerous very degenerated leucocytes. The rete cells are edematous, and there is acanthosis with elongation of the rete pegs. The papillae are markedly edematous.

Hannan and Essex 75.

KERATODERMIA BLENNORRHAGICA.

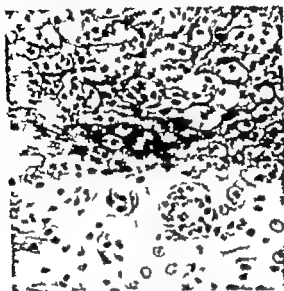


FIG. 114

Keratoderma Blennorrhagica.—This shows completed acroble infiltrated by leukocytes, resting on an endothelial vein. The vein is infiltrated by polymorphs, which at one point have collected to form small intra-epidermal abscesses.
Hematoxylin and Eosin $\times 350$



FIG. 115

Keratoderma Blennorrhagica.—This section shows the most advanced degree of acroble and papillary edema, which is characteristic of the fully developed lesion. The stratum corneum has been lost, and squamous crust rests on the site from which the stratum granulosum is absent.

Hematoxylin and Eosin 50

KERATODERMIA BLENNORRHAGICA.



FIG. 116

Acrotoloma Blennorrhagica — A lesion of longer standing showing desiccating squame crust. This is composed of alternating layers of compacted exudate, collections of degenerated leucocytes, and parakeratotic cells. The stratum granulosum is absent. The acanthosis and papillary edema are subsiding. There is well marked lymphocytic infiltration in the papillary layer of the dermis.

Hematoxylin and Eosin $\times 80$.

PRIMARY VESICULO-BULLOUS ERUPTIONS OF EXTERNAL ORIGIN

SCABIES (Figs 117 120) A vesicle in association with a burrow is the primary skin lesion of scabies. The vesicle often becomes purulent, and the scabies complicated by impetigo and furunculosis.

IMPETIGO CONTAGIOSA (Figs 121 123). A superficially placed intra-epidermal vesicle or bulla is the characteristic lesion of impetigo contagiosa. The roof of the vesicle is usually so thin that the lesion does not remain intact for long, and early rupture is the rule, except on the palms and soles where the stratum corneum is well developed. When the vesicle is preserved the contents, at first clear rapidly become purulent.

Histologically the early lesion consists of a collection of cell-free fluid underneath the stratum corneum. The rete which forms the sides and base of the vesicle shows no degenerative changes in its cells, and intercellular oedema is absent or scanty. There is little or no oedema or cellular reaction in the papillary layer of the dermis. The process by which the bulla develops seems to be purely an exudative one depending on increased capillary filtration, and visible epidermal damage is of the slightest. (See vesicle formation in Eczema section).

While impetigo may be contracted through the contamination of any form of superficial injury there is a close association, particularly in children, between pediculosis capitis (Fig 125) with its attendant puncture wounds and excoriations, and impetigo of the scalp; and in adults, to a less extent, between impetigo of the body and pediculosis corporis (Fig. 124) and phthiriasis pubis (Fig. 128).

SCABIES



FIG. 117
Tick Mite—Sarcoptes Scabiei. Adult female.—Note the
 right legs, with long hair-like processes.
Cleared Preparation 110

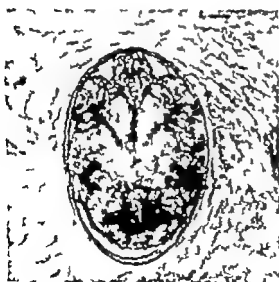


FIG. 118
*Tick Mite—Sarcoptes Scabiei—Embryo still enclosed in
 egg-case.*
Cleared Preparation 350

SCABIES



FIG. 119

*Ick Mite—Sarcoptes Scabiei—Nymph or immature form,
with one leg*

Cleared Preparation 350

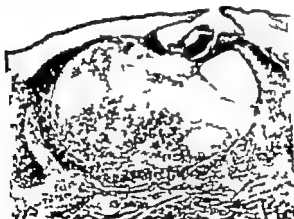


FIG. 120

Scabies. A scabies burrow and the adjacent vesicle.—The parasite has burrowed cavity in the stratum corneum, and an egg is seen lying in this space. Immediately below and to the side of the burrow there is a large unilocular intra-epidermal vesicle the contents of which are slightly purulent. The base of the vesicle is formed by a thin layer of epidermal cells which are being invaded by polymorphs. There is slight vacuolization and perivascular polymorph infiltration in the papillary layer of the dermis. A comparison between the size of the vesicle and the size and number of epidermal cells in its immediate neighborhood suggests that in its formation there must have been considerable loss of epidermal tissue in addition to pushing apart of the cells in the area by an influx of capillary filtrate. (See formation of vesicles in eczema section and Fig. 70).

Hemphill and Evans 80

IMPETIGO CONTAGIOSA.

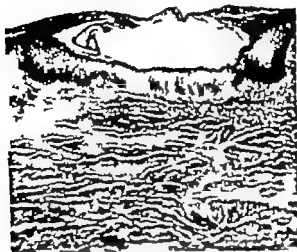


FIG. 121

Impetigo Contagiosa—A bulla has formed beneath the stratum corneum. The contents of the bulla are clear and cell free. The rete which forms its base is stretched and the pegs are absent. There is slight degree of perivascular lymphocytic and leucocytic infiltration, and some separation of the collagen fibres.

Henshaw and Egan $\times 70$

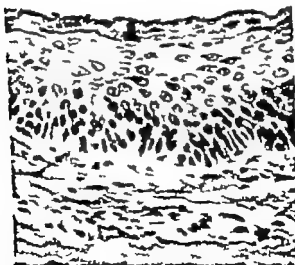


FIG. 122

Impetigo Contagiosa—This shows the structure of the rete forming the base of the bulla. There is slight degree of intercellular oedema and many of the cells stain rather faintly but on the whole the structure is compact and the tissue normal, and there is no evidence of the presence of capillary filtrate to account for the overlying bulla.

Henshaw and Egan $\times 300$

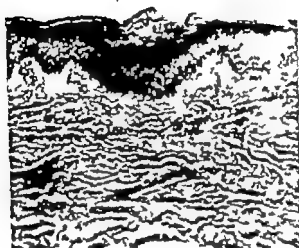


FIG 123

Impetigo Contagiosa — This is an early stage of formation of bulla and appears as separation, by fluid, of the superficial corneal layer of the epidermis.

Hammaker and Ewart x 75

LICE.



FIG. 124

Body Louse—Polioctonus Corporis. Adult female.—The brownish-red coloration in the abdominal area represents the remains of ingested blood and has prevented the clearing of the louse during preparation.

Cleared Preparation $\times 14$



FIG. 125

Head Louse—Polioctonus Capitis. Adult male.

Cleared Preparation 14.



FIG 126

FIG 127

FIG 126—*Nits of Head Louse attached to hair*
Cleared Preparation 14

FIG 127 *Nits of Head Louse attached to hair*—An embryo
 present in one egg case. The other case is empty the
 embryo having escaped.
Cleared Preparation $\times 14$

LICE.



FIG. 128
Cock or Pubic Louse—Phthirus pubis or pubes. Adult.
Cloved Preparation $\times 22$

PUSTULAR ERUPTIONS

A variety of superficial inflammatory skin lesions may become purulent at some period in their evolution, and thus qualify to be included in the category of pustular eruptions. Lesions which are not primarily follicular pustules, but which may become purulent are the bullae and vesicles of *impetigo contagiosa*, all the varieties of herpes, pemphigus, dermatitis herpetiformis, erythema multiforme, the necrotic epidermal lesions of variola and varicella, the vesicles of scabies, and eczema, the papules of rosacea which become necrotic, and the papulo-necrotic tubercles.

The term pustule should however be limited to those inflammatory lesions in which the reaction is from the start a polymorphonuclear leucocytic infiltration of the dermis and epidermis, and if this limitation is observed, it will be seen that the pustular eruptions are confined to superficial and deep inflammations of the hair follicles. The superficial pustule involves the ostium of the follicle, including the epidermis, and the reaction may extend downwards to include the sebaceous gland and the hair root. From the commencement of the reaction there is a brisk infiltration of polymorphonuclear leucocytes in these areas. The lesion is produced by the staphylococcus in all its varieties, and blockage of the ostium, or injury such as might be produced by evulsion of a hair or the lodgement of gritty particles or hydrocarbon products in the ostium, are predisposing factors for its development. Recurrent lesions are liable to develop as a result of autoinoculation of the organism, and pustulation is a common complication of a variety of superficial inflammations either because they lead to scratching and hence to abrasions, or because the staphylococcus finds their discharges a suitable medium for its multiplication and increase in virulence.

The true pustular diseases are SYCOSSIS (Figs. 129-131), ACNE VULGARIS (Figs. 133 and 134), FOLLICULITIS DECALVANS (Fig. 132) and FURUNCULOSIS, and of course single non-recurrent pustules may develop on any area where the hair is strong. A pustule will not develop unless a hair is present in the follicle.

Cutaneous abscesses which simulate spontaneously-arising deep-seated staphylococcal follicular lesions may be caused by biting insects such as TICKS (Fig. 136) and JIGGERS (Fig. 135).

ORF—(Figs. 137-139). This lesion, which is primarily vesicular but which rapidly becomes purulent, occurs on exposed areas, and is usually single. It appears to be contracted as a result of handling sheep suffering from pustular lesions in the region of the hoof. In the human subject a secondary generalised eruption resembling erythema multiforme

may develop during the course of the primary lesion. Neither bacteriological nor serological examination, nor attempts at experimental inoculation have yielded any information as to the cause of the disease, but the presumption is that it is produced by the action of a virus.

The epidermis shows gross disorganisation. There is acanthosis, and large unilocular and multilocular bullae develop. The cells of the rete forming the roof sides, and the base of this area are degenerated. They are swollen the prickles are lost, the cytoplasm is pale, the nuclei stain poorly and may be pyknotic, hydropic vacuoles may be present, in some cells the nuclei have disappeared, the cell membranes are indistinct, and in places clumps of anuclear cells appear to have fused together to form homogeneous hyaline masses. Here and there eosinophil droplets are to be found in the cell bodies.

The papillae show an extreme degree of oedema and the lymph and blood vessels are dilated throughout the entire dermis, in which there is also a diffuse infiltration of inflammatory cells. The lesion resembles those of variola and erythema multiforme, but necrosis is much less in evidence than in the former condition, and the cellular reaction in the dermis, and the epidermal degenerative changes are more marked than in the latter.

STAPHYLOCOCCAL FOLLICULITIS



FIG. 129

Staphylococcal Folliculitis (basal region)—This is a commencing lesion, and evidence of the dermis surrounding the superficial portion of the follicle is more striking than the per-follicular and perivascular infiltrate of leucocytes, which is just commencing.

Henshaw and Essex $\times 45$.



FIG. 130

Staphylococcal Folliculitis. Fully developed pustule (basal region)—A polymorphonuclear leucocytic reaction surrounds the follicle and is not dense at its upper portion where the epidermis has been eroded.

Henshaw and Essex $\times 30$

may develop during the course of the primary lesion. Neither bacteriological nor serological examination, nor attempts at experimental inoculation have yielded any information as to the cause of the disease, but the presumption is that it is produced by the action of a virus.

The epidermis shows gross disorganisation. There is acanthosis, and large unilocular and multilocular bullae develop. The cells of the rete forming the roof sides and the base of this area are degenerated. They are swollen, the prickles are lost, the cytoplasm is pale, the nuclei stain poorly and may be pyknotic, hydropic vacuoles may be present, in some cells the nuclei have disappeared, the cell membranes are indistinct, and in places clumps of anuclear cells appear to have fused together to form homogeneous hyaline masses. Here and there eosinophil droplets are to be found in the cell bodies.

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STAPHYLOCOCCAL FOLLICULITIS

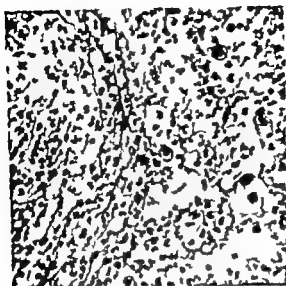


FIG. 131

Staphylococcal Folliculitis—An area from Fig. 130 showing the dense polymorphonuclear infiltration, diminished epithelial cells, and debris.

Hamman and Egan 575



FIG. 132

Folliculitis Decalvans—There is dense polymorphonuclear peri-follicular infiltration surrounding the entire length of the hair follicle which is destroyed by the process. In the adjacent dermis perivascular leucocytic infiltration is present.

Hamman and Egan 40



FIG. 133

Acne vulgaris, Comedo stage.—This shows the keratinized mass of horny cells and sequestered sebacea plugging the orifice of hair follicle. The follicular epithelium is thinned due to pressure. In the surrounding dermis the vessels are dilated and there are number of lymphocytes scattered through it.

Hamacher and Essex x 45.



FIG. 134

Pustular Acne.—A plugged follicle surrounded by dense polymorphonuclear infiltration.

Hamacher and Essex x 30.

JIGGER FLEA AND TICK



FIG. 135
Jigger or Sand Flea—
Sarcophaga (or *Phlebotomus*)
Cleared Preparation 50.

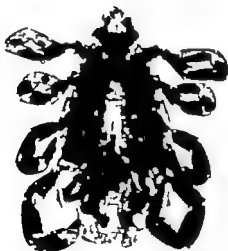


FIG. 136
Tick—*Dermacentor undecimnotatus*. Adult.
Cleared Preparation 20.



FIG. 137

Orf.—Numerous multilocular and unilocular vesicles are present in the middle of the epidermis. The epidermis is deepened and the individual cells are swollen and degenerated. There is marked papillary edema and communicating channels exist between this edematous area and the intra-epidermal collection of fluid. The dermis is edematous and infiltrated by inflammatory cells.

Hamahan and East $\times 15$.



FIG. 138

Orf.—There is intra-epidermal and sub-epidermal collection of acridin, and they are interconnected. The epidermal cells are swollen and degenerated. There is diffuse cellular infiltration of polymorphs and lymphocytes in the dermis.

Hamahan and East $\times 30$.



FIG. 139

Orf.—The rete cells are swollen, the cytoplasm stains poorly, adjacent cells are condensing and their cell membranes are indistinct, some are becoming completely disintegrated, and many are anuclear. Pyknosis is to be seen and in one cell three eosinophilic lysolene droplets are present in the cell cytoplasm.

Hamahan and East $\times 450$.

TINEA.

(Figs. 140-144.)

Tinea corporis lesions present a variable histological picture depending on the virulence of the fungus and the site affected. The changes range from a slight degree of simple inflammation, to vesiculation of the eczema type, and follicular pustulation comparable to that seen in sycosis.

In *tinea kerion*, in which the tissues have become allergic to the fungus toxin with the subsequent development of an immunity to the organism, there is a deep-seated perifollicular reaction of granulomatous type, with lymphocytes, plasma cells, histiocytes, and an occasional giant cell. There may be some suppuration and as the reaction subsides scar tissue develops.

TINEA CORPORIS AND TINEA KERION

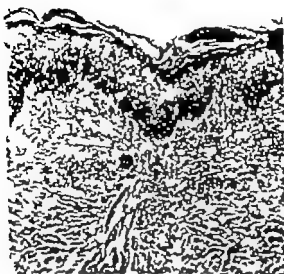


FIG. 140

Tinea Corporis—Hyperkeratosis and parakeratosis are present, and there is some acanthosis which is flattened with papillary cords and perivascular lymphocyte infiltration has flattened the rete pegs. The appearance is that of simple eczematization, and presents no individual features. *Henshaw and Egan* $\times 65$.



FIG. 141

Tinea Kerion—There is deep-seated and widespread perivascular inflammatory reaction. *Henshaw and Egan* 35



FIG. 142

Tinea Kerion—There is intense and cellular infiltration composed of lymphocytes, few histiocytes and an occasional polymorphonuclear leucocyte. *Henshaw and Egan* 370.

ROSACEA AND RHINOPHYMA.

(Fig. 143)

Although Rosacea is an erythematopapular eruption and not a true primary pustular disease, the papules so frequently become necrotic and develop into virtual pustules that it may be most conveniently considered along with the pustular diseases.

In Rosacea there is a functional derangement of the cutaneous vessels of the face which is first noticed as frequent and repeated flushing of the cheeks, forehead, chin, and nose, and later by permanent dilatation of a number of these vessels. Associated with this vascular instability there is a tendency to the formation of indolent papules and pustules, and later a slow hypertrophy of the sebaceous glands may develop on the affected areas. The hypertrophy is most marked on the nose, where it may reach considerable proportions and form fleshy nodules. When the sebaceous gland hyperplasia has reached such a degree as to produce a noticeable tumour the clinical condition is known as Rhinophyma. The overgrowth of sebaceous gland tissue is a pure hyperplasia and is not adenomatous in character. The inflammatory features of the disease are continued in the vicinity of the hyperplastic glands, and the repeated congestion associated with them predisposes to further glandular hyperplasia.

GRANULOMA PYOGENICUM.

(Fig 144)

This condition is essentially a hypertrophy of granulation tissue following injury usually a superficial puncture wound. The granulation tissue has an epidermal covering which may be acanthotic in some places, and exceedingly thin in others. The newly formed fibrous structure contains numerous capillaries, fibroblasts, and histiocytes, and lymphocytes are abundantly scattered throughout.

ROSACEA AND RHINOPHYMA.

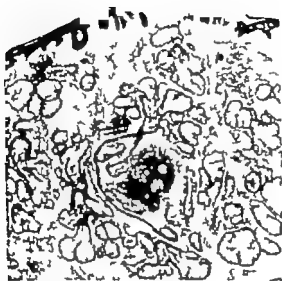


FIG. 143

Rosacea and Rhinophyma.—This section shows hypertrophy and hyperplasia of the sebaceous glands. The gland structure normal and there is no evidence of cystic. The lesion is hyperplastic and not truly adenomatous. A chronic inflammatory reaction is present in the immediate vicinity of sebaceous gland. (Compare with Nerve Sebacea, Fig. 260)

Hershey and Egan $\times 15$

GRANULOMA PYOGENICUM.



FIG. 144

Granuloma Pyogenicum.—The epidermis is acanthotic, and there are some parakeratosis. The dermis contains many newly formed capillaries, and there are numerous histiocytes and lymphocytes scattered throughout the somewhat homogeneous collagen.

Henshaw and Egan 60.

TRAUMATIC INJURIES AND THE EFFECTS OF STRESS

The initial effect of trauma on the skin is a variable degree of destruction of cutaneous tissue, the extent of which depends on the nature and severity of the causative agent which may also determine the occurrence of special sequelae. The common feature in all injuries is a simple inflammatory reaction, intensified if pyogenic infection has supervened, followed by the development of granulation tissue and finally scar tissue.

BURNS (Figs. 145-147). The lesions produced by burning range from a slight erythema to complete charring of the skin and subcutaneous tissues. In scalding, e.g. by water or steam, vesication may be a feature. The histological changes therefore, vary from slight oedema of the epidermis with congestion of the dermis and a minimal leucocytic infiltration, to gross necrosis of the whole skin. In the latter case the necrotic area becomes saturated with serous exudate and there is intense congestion and leucocytic infiltration beneath it (Fig. 147). Later as healing occurs a fibrous scar tissue develops, and in the more severe and deeper burns a hypertrophic scar or a keloid may be produced. Secondary infection of the devitalised tissue intensifies the inflammatory reaction and leads to excessive scarring.

WOUNDS (Figs. 148-150). In simple physical trauma the changes are those of simple inflammation and as healing occurs the amount of granulation tissue and subsequent scarring is determined by the degree of tissue destruction or of associated infection.

Foreign bodies may be driven into the wound and complicate the picture. Thus particles such as copying ink, iron, dust, or silica may be introduced, and foreign-body giant cells form around them. If the particles are abundant, and especially if they are insoluble or nearly so, they may remain in the tissues for long periods. Particles of silica excite considerable fibrosis in the surrounding tissues and may give rise to the formation of foreign body giant cells and nodules closely resembling the tubercles of tuberculosis. Silica particles embedded in the dermis can usually be seen in the inflammatory or fibrous nodules which they excite—they are doubly refractile and can be displayed by examination with polarised light. Iron particles (Figs. 148 and 149), may be recognised by their chemical reactions. Copying ink (Fig. 150), deserves special mention as its introduction into the tissues may cause considerable necrosis and an intense inflammatory reaction.

VARICOSE ULCER (Figs. 151 and 152). Varicosity of the cutaneous venules of the leg predisposes to the development of ulceration in the lower third of the limb. This may be due to external injury of the poorly nourished tissue with subsequent necrosis due to mild infection,

or it may result from thrombosis. Whatever the mechanism a localised gangrene involving the entire skin and sometimes the subcutaneous tissue is the result. Ulceration may be accompanied, or preceded by eczema.

The CORN (Fig. 153) and BUNION (Figs. 154 and 155) are both reactions to long continued or intermittent stress. In the corn there is a localised dense hyperkeratosis which not only projects on to the surface, but presses on the underlying rete causing it to become thinned and stretched and to lose many of its pegs. The subjacent dermis shows a varying degree of chronic inflammatory reaction. The bunion shows an area of degenerated, glassy and swollen collagen fibres, surrounded by chronic inflammation and covered by a hyperkeratotic epidermis.

SCAR and KELOID (Figs. 156 and 157). The scar which results from the organisation of granulation tissue presents a thinned, sometimes hyperkeratotic epidermis with a complete absence of rete pegs. In the dermis the collagen fibres are increased in density and deficient in connective tissue cells, and the elastic fibres are entirely absent or much reduced in quantity although they tend to be concentrated in the immediately adjacent surrounding skin. Sweat glands, hair follicles and sebaceous glands are absent. In scars of recent origin there may be a varying amount of perivascular lymphocytic infiltration and an excess of swollen, cellular recently formed collagen. At this stage the scar may appear hypertrophic but the infiltration later disappears and the collagen contracts and ceases to be swollen. In some cases, however and for no known reason, the newly formed collagen and fibroblasts continue to develop. A hypertrophic mass which may greatly exceed the area of the original injury and which shows no subsequent contraction is thus produced. This condition is known as Keloid, and from its microscopic appearance and its tendency to recur it bears some relationship to tumour formation.

STUMP NEUROMA (Fig. 158). The condition known as stump neuroma consists of a mass of scar tissue formed round the end of a divided nerve from which numerous nerve fibrils arise and ramify throughout the scar.

BURNS



FIG. 145

Burn, 2nd Degree—The epidermis is destroyed and is represented by a collection of dead cells and serum fluid. The superficial layers of the dermis are markedly disorganized.
Hammaker and Egan $\times 16$.

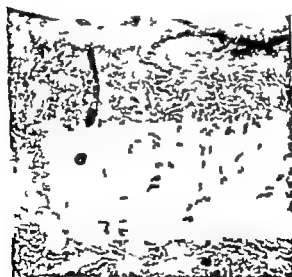


FIG. 146

Burn, 3rd Degree—A higher power view of the edge of the burn shown in Fig. 145. Here the epidermis is gone and small clusters of cells are seen in the deeper layers of the dermis.

Hammaker and Egan 50



FIG. 147

Burn, 3rd Degree.—The epidermis is destroyed and replaced by
consolidated coagulum. The superficial part of the dermis is necrosed,
and there is cellular debris. Each extends into the deeper part of the dermis.

Hematoxylin and Eosin $\times 10$.

WOUNDS



FIG. 148

Wound due to iron filings driven into skin—The wound has become chronic and iron pigment can be seen in the surface exudate and deep in the wound. There is considerable fibrosis around.

Hematoxylin and Eosin $\times 8$.



FIG. 149

The same wound as Fig. 148—The section has been treated by HCl and Potassium ferricyanide, which turns the iron deposit deep blue.

Prussian Blue Reaction conventional Fuchsin $\times 8$.

FOREIGN BODIES

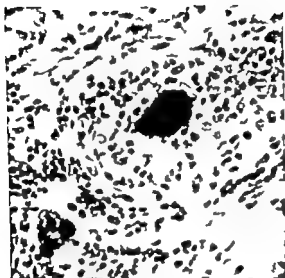


FIG. 150

Carpenter Ink deposit—The particles of ink (accidentally introduced through the skin) lie in the dermis and are surrounded by large pale-staining histiocytes, some of which have foamed around the larger deposits, forming "foreign body giant cells." Scattered lymphocytes are present and towards the periphery there is fibrinolytic reaction.

Hammann and Evans 375.

VARICOSE ULCER.



FIG. 151

Varicose Ulcer—The section shows an acanthotic epidermis which slopes steeply at the edge of the ulcer. The underlying dermis is slightly oedematous, the vessels are dilated, and there is moderate leucocytic infiltration. Between the dermis proper and the purulent exudate on the left of the section which constitutes the surface of the ulcer, there is an area of recent granulation tissue containing numerous newly formed vessels and fibroblasts. *Harrison and Egan* $\times 15$



FIG. 152

Varicose Ulcer—T the left of the section the epidermis above, there is purulent exudate in the upper portion and beneath this an area of recent granulation tissue. T the right of the section the epidermis is intact the subepidermal dermis is slightly oedematous, more cellular than normal, its vessels are dilated, and there is moderate leucocytic infiltration. *Harrison and Egan* 60



FIG. 153

Class—"Corn"—From foot—There is circumscribed excessive keratinization forming mass of horny material which is pressed on to the underlying epidermis. This is hyperplastic but is stretched by the pressure of the horny mass.

Ivan Hammett and Van Genn 24

BUNION

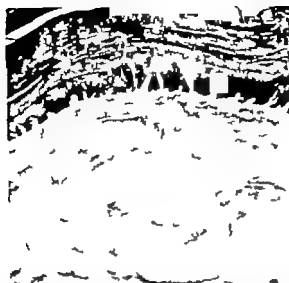


FIG. 134

Bunion of toe—1. the epidermis there is excessive cornification and thick rete pegs. Immediately beneath are many dilated vessels. In the dermis is large ill-defined mass of dense scar tissue composed of collagen the fibres of which run more or less in concentric fashion. Many small blood vessels are present in the peripheral layers with collections of chronic inflammatory cells around them.

Hamman and Egan 14



FIG. 135

Bunion of toe—A higher power view of the margin of the fibrous mass, showing the collagen strands and several small vessels. The homogeneous area in the central part composed of more hyaline collagen, with few nuclei.

Hamman and Egan 65

SCAR AND KELOID

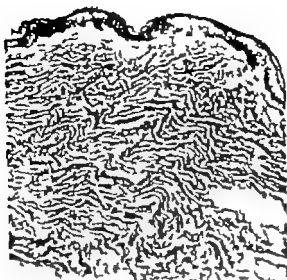


FIG. 156

An area of old scar tissue.—The epidermis is of normal thickness but the rete pegs are scanty and small. Underneath the epidermis there is a deep layer of more or less parallel wavy collagen fibres. The connective tissue cells are greatly reduced in number. There are few blood vessels, as the scar is old. Elastic tissue is absent.

Hematoxylin and Eosin $\times 60$.

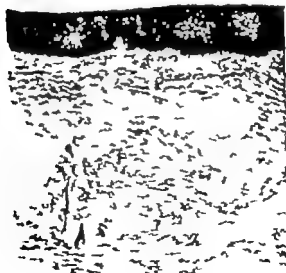


FIG. 157

A keloid.—This is an overgrowth of scar tissue, and consists of great excess of collagen bundles with quite numerous fibroblasts and few scattered lymphocytes.

Hematoxylin and Eosin $\times 60$.

"STUMP NEUROMA."

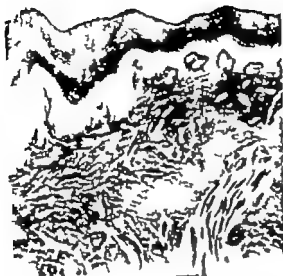


FIG 158

"Stump neuroma. — This is not true tumor as its current more suggests, but mass of scar tissue permeated by proliferating bundles of nerve fibres, stained black. It may form nodule of nerve size at the end of the severed nerve. This specimen is covered with thick layer of epidermis. This is an old lesion, showing few remaining blood vessels.

Hobbs' *Silver Method and Contents* 35

THE RETICULOSES

The conditions included under this term have as their common feature a progressive hyperplasia of more or less undifferentiated mesenchymal cells. Actual tumour formation, with destruction and invasion of normal tissues, may also develop from these cells, giving rise to the various forms of reticulo- or reticulum-celled sarcoma.

The varieties of reticulosis were originally described in relation to certain hyperplastic changes in lymph glands, but the cells concerned have a wider distribution in the body. Normally they are found in the lymph glands, the spleen, and bone marrow where some of them form the reticulum or parenchyma, others are lining cells of the sinuses, and yet others are applied to the branching fibrils of the reticular framework of these tissues. They also occur as undifferentiated mesenchymal cells lying in the meshes of connective tissues, including the dermis. When they undergo hyperplasia they may form groups of individual polygonal cells, syncytial masses, or even giant cells, and they may also differentiate in various directions giving rise to histiocytes, fibrocytes, lymphocytes, and hæmic cells, especially of the myeloid series. A single type or a mixture of these various cells may be found as the result of the hyperplastic process. Some of the cells produce fine argyrophil fibrils of reticulum.

A hyperplasia of the reticular cells and their derivatives may form part of a number of inflammations of known origin—e.g. cutaneous tuberculosis—the process being referred to as an inflammatory reticulosis. A progressive hyperplasia may also occur as a result of unknown causes, as in certain "lymphadenopathies," and it is to this latter group that the term *Reticulosis* is restricted. The cutaneous manifestations of reticulosis are as follows—lymphadenoma cutis, mycosis fungoides, leukaemia cutis—the cells of the leukaemias representing hyperplastic derivatives of the undifferentiated marrow reticular cells, the monocytic variety being perhaps the most closely related to the original reticular cell—and reticulum-celled sarcoma.

In LYMPHADENOMA, or Hodgkin's disease (Figs. 159, 160 and 161), the superficial nodules have the same general characteristics as those in the lymph glands and spleen. There is a mixture of reticular cells, including histiocytes, recognisable as large polygonal cells with round or indented nuclei, giant cells (originally described by Greenfield and later by Sternberg and by Dorothy Reed), together with variable numbers of eosinophils and fibroblasts. A delicate fibrillary meshwork of argyrophil fibrils is present amongst the cells, and later it is replaced by collagen fibres.

MYCOSIS FUNGOIDES (Figs 162, 163 and 164) also presents a variety of types of reticular cells which infiltrate and form masses in the superficial dermis. These cells comprise histiocytes, some of which are multinucleated, lymphocytes, and plasma cells, enmeshed in a fine reticulum.

LEUKAEMIA (Figs 165 166 and 167). Skin involvement is common in lymphatic and monocytic leukaemia, less so in the myeloid variety. In all three varieties there is present in the upper and middle dermis an infiltration of cells which are more or less uniform in type and characteristic for the particular disease, and which have accumulated as a result of local hyperplasia, or by infiltration from the capillaries.

RETICULO-SARCOMA (Figs 168-174) In this case the hyperplasia is malignant in character with invasion and destruction of the surrounding stroma and spreads by the blood stream and lymphatics. There is general uniformity in the type of cell present in a given tumour but any variant of reticular cell may be involved. A fine reticulum is usually present throughout the cell masses. A sarcoma may arise primarily in the dermis, either from a single site or from several points simultaneously or metastases from a distant site may be widely deposited in the skin.

LYMPHADENOMA.

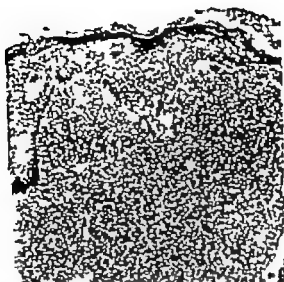


FIG. 159

Lymphadenoma (Hodgkin Disease).—Capsule visible. From the under surface of the epidermis and extending throughout the entire dermis is heavy infiltration by many cells amongst which can be distinguished several larger cells.
Hermann and Egan, × 75.

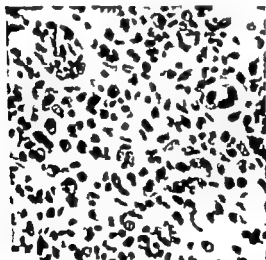


FIG. 160

Lymphadenoma.—This shows the cells of the nodule in detail. One of the large Hodgkin (so-called Dorothy Reed) cells present near the margin is a small giant cell with several nuclei; many other histiocyte-type cells having large oval nuclei are distributed irregularly amongst many lymphocytes, plasma cells, and some eosinophils.
Hermann and Egan, × 450.

LYMPHADENOMA.

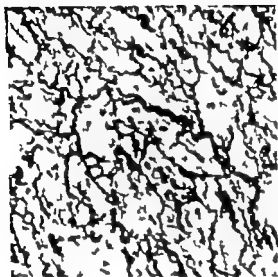


FIG. 161

Lymphadenoma.—A fine reticulum of delicate fibrils present between groups of the cells and even between and surrounding individual cells.

Few Method, Counterstained Fuchsin 475

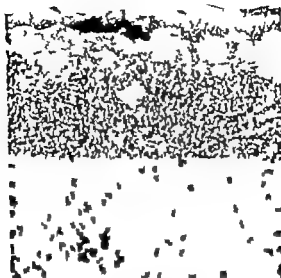


FIG. 162

Mycosis Fungoides.—In the dermis there is dense infiltration with great numbers of cells of various types. Several small vessels run amongst the infiltrating cells.
Hematoxylin and Eosin $\times 75$.

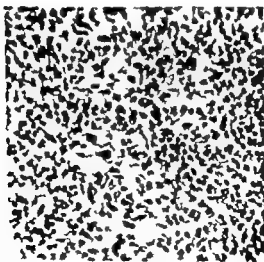


FIG. 163

Mycosis Fungoides.—A portion of the nodules in more detail. The cells are chiefly of the histiocytic type—large, polygonal, with rather foamy staining nuclei. Interspersed with these are scattered lymphocytes.
Hematoxylin and Eosin $\times 350$.

MYCOSIS FUNGOIDES



FIG. 164

Mycosis Fungoides.—There is reticulation in the form of fine dark threads intimately related to the cells.
Foot Method Constantineau Fuchsin $\times 400$.

MONOCYTIC LEUKAEMIA.



FIG. 165

Monocytic Leukemia—*Cutaneous nodules*. This general view shows the upper dermis extensively infiltrated with cells.
Hamaker and Evans 25



FIG. 166

Monocytic Leukemia—The same as Fig. 165 to show the cellular infiltration, partly in the vessels of the tissue of the dermis but more prominently in the vessels.
Hamaker and Evans 70.

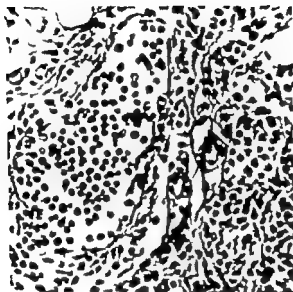


FIG. 167

Monocytic Leukaemia. The same as Fig. 165.—Here is shown small blood vessel distended by great numbers of large monocytes and relatively few red corpuscles. Smaller cells lie in the meshes of the adjacent connective tissue.

Hassall and Egan × 275.

SARCOMA.

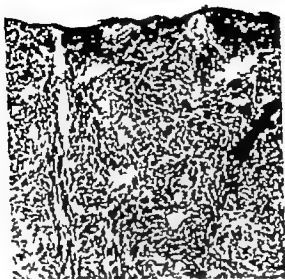


FIG. 168

Cutaneous Sarcoma, large spindle-celled type. From Forehead of an infant.—The tumour composed of masses and strands of large spindle cells amongst which are several thin-walled, poorly supported blood vessels. The tumour cells have crested through the epidermis.

Hamman and Enns X 85.



FIG. 169

Cutaneous Sarcoma, large spindle-celled type. Same case as in Fig. 168.—There is recurrence following removal of the original tumour. The cells have the same characters and are infiltrating widely into the epidermis and dermis. The margin poorly defined.

Hamman and Enns 43.

SARCOMA



FIG. 170

Coloured Sarcoma, large spindle-celled type. From same case as Figs 168 & 169.—The reticulum is stained and appears as coarse filaments between groups of the large spindle cells and as finer threads between individual cells, though some cells show no reticulum around them.

Fast Method, Counterstained Fuchsin. 475.

SARCOMA.



FIG. 171

Cutaneous Metastases of Sarcoma—This view merely shows the general cellular picture of the tumour both infiltrating the dermis and stretching the epidermis. The margin is ill defined.
Hermann and Egan 11

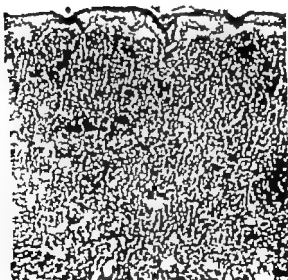


FIG. 172

Cutaneous Metastases of Sarcoma—Same as Fig. 171—The cellular character of the nodules better seen here, the normal dermis being quite obscured and replaced by the tumour tissue.
Hermann and Egan 10

SARCOMA.

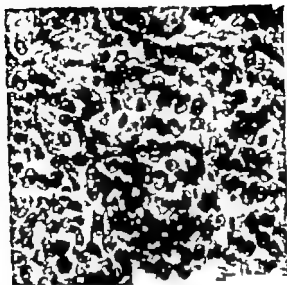


FIG. 173

Colossal Metastasis of Sarcoma. Same as Fig. 171.—The cells are all large, rounded or polyhedral in shape, with large pale-staining ovoid nuclei—several mitoses are seen. This shows the sarcoma terribly as round-celled type.

Hematoxylin and Eosin $\times 490$

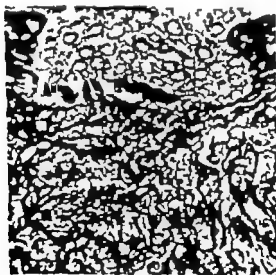


FIG. 174

Colossal Metastasis of Sarcoma. Same as Fig. 171.—With this staining method there is seen definite formation of reticulum fibers around the cells of the tumor indicating its character as reticulum-celled sarcoma.

Fast Method Counterstained Fuchsin 475

GRANULOMATOUS CONDITIONS

Under this heading are included and illustrated the following — syphilis, lupus, tuberculides and sarcoid, actinomycosis, cutaneous Leishmaniasis (oriental sore). They all have the common feature of a cellular infiltration of the tissues by the various types of inflammatory cells and the formation of granulation tissue, often in excessive amount, with the final development of fibrosis. While the histological picture may be characteristic, e.g. in lupus, a closely similar appearance may be presented by syphilis. Frequently it is necessary to have some confirmatory evidence, such as is given by special staining methods, to demonstrate the causal organism, before a positive diagnosis can be made.

SYPHILIS. The various cutaneous manifestations of syphilis all have the same general type of histological picture varying only in the amount of oedema or cellular infiltration which is present. This applies especially to the primary and secondary types of lesion. The tertiary lesion, e.g. the gumma, presents a much greater degree of fibroblastic proliferation, often with necrosis in its centre, but the vascular changes and cellular infiltration are similar to those of the other stages.

The Primary Chancere (Figs. 175-178) shows a dense infiltration of the superficial dermis by mononuclear cells, largely composed of plasma cells along with a variable number of histiocytes and lymphocytes. The capillary vessels are increased in number they may exhibit endothelial swelling and proliferation, and are often surrounded by a collar of these infiltrating cells (Fig. 178). The epidermis is oedematous, infiltrated by mononuclears, and in due course ulcerates. Spirochaetes are present in great numbers in this lesion, or in a smear made from it, but they are only demonstrable by special staining methods (Figs. 179 and 180).

In the *Papulo-squamous* (Fig. 181), and *Condylomatous lesions* (Fig. 182), the above features are present in varying degree. The condyloma is a papulo-squamous lesion in which an epidermal hyperplasia has occurred on account of the moist situation of the lesion. In the later manifestations, the *Nodulo-cutaneous* syphilide (Figs. 183-186), and the *Gumma* (Figs. 187-189), there is fibroblastic proliferation as well as cellular infiltration in the nodules, and the inflammatory process extends more deeply and widely in the dermis. In these late lesions spirochaetes are scanty or absent.

When the different cutaneous manifestations of syphilitic infection are examined as a whole and contrasted one with the other it will be seen that the cellular reaction evoked during the active phases is essentially uniform, and differs only in the amount and depth of the infiltration, and not in the type of cell. The clinical lesions characteristic of the various

stages of the infection differ from each other much more obviously than do the histological findings. These variations in the distribution, configuration, and depth of the successive eruptive elements, together with the intervening periods of quiescence, suggest that throughout the course of the disease there is a progressive modification in the immunological and allergic processes stimulated by the infection.

LUPUS (Figs. 190-193) This is a definite tuberculous lesion which is characterised by typical tubercles, with "endotheloid" cells and giant cells, which coalesce and are diffusely distributed throughout the superficial dermis. Caseation is seldom a prominent feature. There is considerable fibroblastic reaction around the tubercles, the amount depending on the age and activity of the lesion. In the verrucose form there is marked hyperplasia and hyperkeratosis of the epidermis. Ulceration of the surface may occur either as a result of secondary pyogenic infection of the surface, or following necrosis of underlying cartilage, e.g. of the nose, which has become invaded by the tuberculous process.

TUBERCULIDES (Figs. 194 and 195), have similar histological features to those of lupus, but in them the tubercles are smaller and much more discrete and clearly demarcated from the surrounding dermis, in which they are deeply placed.

In the papulo-necrotic form necrosis of the tissue overlying the inflammatory nodules is prone to occur with the formation of superficial ulcers.

SARCOID (Figs. 196 and 197) The histological picture in the various clinical types of sarcoid is essentially that of a tuberculous process. The infiltration is circumscribed, it is deeply situated in the dermis, and caseation is absent.

Lupus, the tuberculides, and sarcoid are histologically very similar varying in degree rather than in kind and tubercle bacilli are rarely demonstrable even in a frank lupus. Sensitivity to tuberculin varies in the three groups, lupus cases showing a higher percentage of positives than "normal," the tuberculides a slightly lower percentage, and the sarcoid group a very low rate. It may be that variations in sensitivity to tuberculin account for the clinical and histological variations in the three conditions.

LEPROSY (Figs. 198-201) The cutaneous nodules are formed by a heavy infiltration of the dermis by large mononuclear cells (histiocytes) many of which are filled with *Lepra* bacilli, demonstrable by a modified Ziehl-Neelsen staining (Fig. 201). There is also some fibroblastic proliferation and oedema, and later fibrosis occurs. Necrosis in the infiltrated area may lead to ulceration. Sometimes these cellular infiltrations are present along small superficial nerve bundles (Fig. 198), a distribution which is also seen in the deep nerves in the anaesthetic form of leprosy.

ACTINOMYCOSIS (Figs. 202 and 203) In the skin this usually takes the form of a sinus leading from an subjacent focus. The sinus is lined by granulation tissue, which at the orifice is covered with epithelium. Pus lies in the lumen of the sinus or covers its walls, and in it are found the felted masses of the mycelium of *Streptothrix Actinomyces*, stainable by Gram's method (Fig. 203). The lesion is that of a chronic inflammation and only the finding of the casual organism gives the clue to the diagnosis.

CUTANEOUS LEISHMANIASIS (Figs. 205-210), occurs as a chronic indolent nodule or plaque in the skin, which may ulcerate, and which is caused by an infection of Leishman-Donovan bodies. These may be found free or in mononuclear phagocytes in scrapings from, or in sections of the sore (Figs. 209 and 210). They are small bi-nucleated bodies easily demonstrated by Leishman's or Giemsa's stains. The lesion in the skin is chronic inflammatory without specific characters, sometimes even showing nodules simulating cellular tubercles (Fig. 207). The disease is transmitted by the sandfly (*Phlebotomus papatasi*) (Fig. 204).

RHINOSCLEROMA (Fig. 211) is a chronic granulomatous and sclerosing condition which affects the nose and adjacent structures. Histologically a dense infiltration of plasma cells and large vacuolated histiocytes is present in the middle and lower dermis and may extend to involve the subcutaneous tissues. The short encapsulated bacillus of rhinoscleroma is present in clumps within the large histiocytes, and it can easily be demonstrated in smears from the lesion.

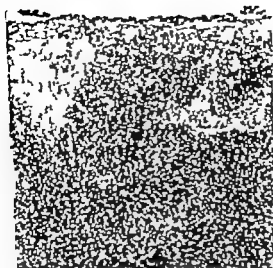


FIG. 175

Primary Syphilitic Sores (Chancres), general view of surface—The epidermis, which is still intact though thinned, is being infiltrated by mononuclear cells; leukocytes are present in great numbers diffusely throughout the dermis; amongst them run many capillaries.
Hematoxylin and Eosin $\times 75$

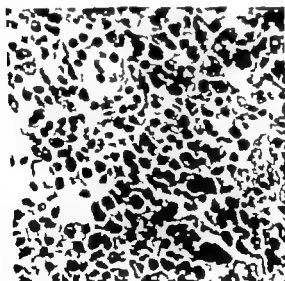


FIG. 176

Primary Sores A portion of Fig. 175 to show details of cells—There are many various mononuclear types. Plasma cells with eccentric deeply-staining nuclei are numerous, amongst them are polymorphous leukocytes with round or oval pale-staining nuclei; lymphocytes, with small dark nuclei, and an occasional polymorphous leukocyte completes the collection.

Hematoxylin and Eosin $\times 500$



FIG. 177

Primary Sore. A slightly eddy lesion.—The epidermis is elevated towards one side and infiltrated by mononuclear cells elsewhere. The infiltration of the dermis is not so dense as in Fig 175, it is edematous, and there are many small blood vessels surrounded by thick collar of mononuclear cells.

Hematoxylin and Eosin $\times 75$.

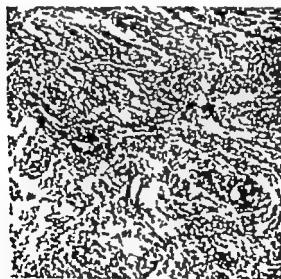


FIG. 178

Primary Sore. to show especially the small vessels.—Several of these are seen with their walls and the perivascular area infiltrated by mononuclear cells; there is also some swelling of the vascular endothelium. There is edema of the adjacent stroma.

Hematoxylin and Eosin 130

SYPHILIS

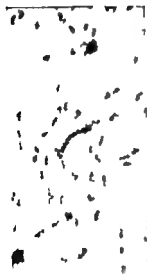


FIG. 179

FIG. 179 *Traperona Pallidum*—The appearance of syphilis as seen in smears from primary sore.
Stained by Fasten method 1000

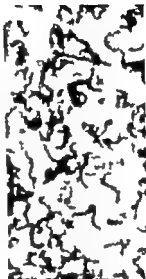


FIG. 180

FIG. 180 *Traperona Pallidum*—As seen in the tissue.
Stained by Dufell method 1010

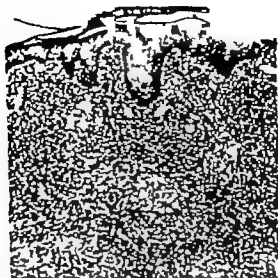


FIG. 181

Pyoderma-gumma Siphilide.—The infiltration of the dermis has the same characters as already described and shown in the primary zone (see Figs 175 and 177), but the epidermis here intact and slightly hyperkeratotic.

Hamaker and Essex 75.



FIG. 182

Carcinoma latens.—Carcinoma latens in pyoderma-gumma lesion which has become hypertrophied owing to its peri-oral, vulvar or scrotal site. There is much hyperplasia of the epidermis with exaggeration of the rete pegs. In the dermis is the same type of cellular infiltration as that seen in other cutaneous nodules (see Figs 185 & 186).

Hamaker and Essex 95.

SYPHILIS



FIG. 183

Nodulo-cystic Syphilis with ulceration.—The infiltration of the dermis is similar to that described in Figs 175, 177 and 181 but the epidermis is involved by the mononuclear infiltration and has ulcerated. There is hyperkeratosis over the ulcerated area. In the remaining part of the epidermis there is hyperplasia. *Hematoxylin and Eosin* $\times 75$.

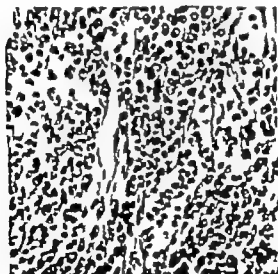


FIG. 184

Nodulo-cystic Syphilis.—A portion of the preceding figure (Fig. 183) to show details of the cellular infiltration. The majority of the cells are plasma cells, intermingled with lymphocytes, lymphocytes and an occasional polymorph leucocyte. Several capillary blood vessels are seen; they exhibit prominence of their endothelial cells and two contain many polymorph leucocytes in their lumina. *Hematoxylin and Eosin* $\times 350$.

SYPHILIS



FIG. 185

Nodule-cutaneous Syphilis.—In this example the cellular infiltration is definitely aggregated around the small blood vessels both in the deeper parts of the dermis and in the papillae. There is epidermal hyperplasia, with prolongation of the rete pegs and slight hyperkeratosis.

Hemmes and Essex $\times 70$.

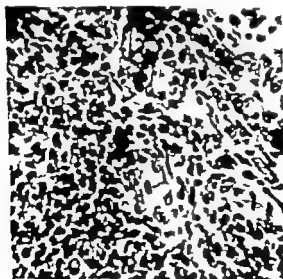


FIG. 186

Nodule-cutaneous Syphilis, part of Fig. 185 to show details.—Several small vessels are shown their endothelium is swollen and proliferated and there is dense infiltration around them, the cells being mainly plasma cells and histiocytes.

Hemmes and Essex 375.



FIG. 187

Cornua of skin, parietal skin.—This shows merely an extensive infiltration of the dermis area by granulomatous nodules.
Hematoxylin and Eosin $\times 12$.



FIG. 188

Cornua of skin, portion of the preceding figure, FIG. 187.—The dermis is heavily infiltrated with mononuclear cells, in places definitely aggregated around small blood vessels; there is also fibroblastic increase.
Hematoxylin and Eosin $\times 60$.

SYPHILIS

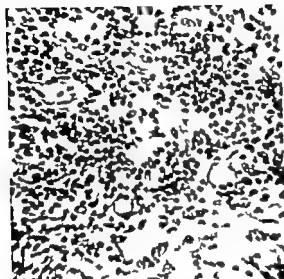


FIG. 189

Course of it is in more detail—Several small vessels are seen, in some of which the lumen is almost obliterated by swelling and proliferation of the endothelium. In and around these vessels there is an infiltration of mononuclear cells. A few fibrin casts are seen around the vessels and in the clearer areas.

Harrison and Egan 330

LUPUS VULGARIS

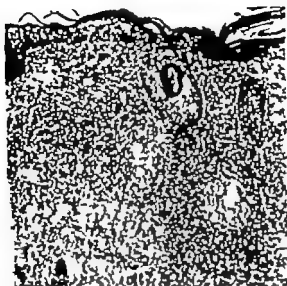


FIG. 190

Lupus Vulgaris—There is slight hyperkeratosis with thinning of the epidermis and substitution of it by lymphocytes. In the dermis there is diffuse infiltration by cells, mainly lymphocytes, and in its deeper area there are several tuberculous nodules composed of large pale-staining histiocytes (endothelial cells).

Hermann and Egan 95



FIG. 191

Collected Lupus—A variant of *Lupus Vulgaris*—The tubercles and diffuse lymphocyte infiltration of the dermis are of the same character as those already described in Fig. 190. There is parakeratosis and also slight intercellular edema of the epidermis.

Hermann and Egan 75

LUPUS VERRUCOSUS

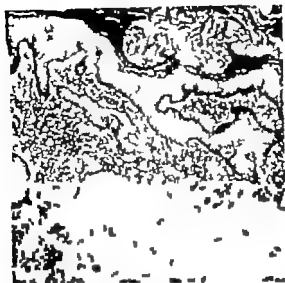


FIG. 192

Lupus Verrucosus. A variant of *Lupus Vulgaris*.—Here there is much hyperplasia of the epidermis with extensive necrosis. In the deeper the tuberculous lesion has the same general characters as in Fig. 190. In this specimen giant cells are numerous in the tuberculous nodules.

Hamman and Eason 50

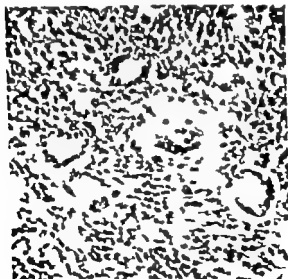


FIG. 193

Lupus Verrucosus. A portion of Fig. 192.—This is to show the details of the tuberculous nodule. Several giant cells are seen with surrounding histiocytes and lymphocytes.

Hamman and Eason 275

TUBERCULIDE.



FIG. 194

Papulo-Necrotic Tuberculide, from the head, general view—Scattered through the dermis and extending as deeply as the subcutis but are granulomatous nodules which vary in size, and are clearly demarcated from the surrounding tissue.

Hemadon and Eason $\times 15$.



FIG. 195

Tuberculide, more detailed view—A typical giant cell is seen surrounded by histiocytes with lymphocytes further out.

Hemadon and Eason $\times 225$.

SARCOIDOSIS



FIG. 196

Sarcoidosis, subcutaneous nodules.—Several granulomatous nodules, resembling tubercles, in deep in the dermis.
Harrison and Egan 45



FIG. 197

Sarcoidosis. *T* shows details of the nodule.—This consists mainly of large histiocytes with an occasional small pyknotic cell. Cells around are scattered lymphocytes. The margins of the nodule clear cut. There is no caseation. This lesion bears close resemblance to tuberculous nodules.
Harrison and Egan 110.

LEPROSY



FIG. 198

Leprosy, early tubercled form.—The granulomatous nodules follow the distribution of the small cutaneous nerves, one of which is seen cut obliquely in the lower left-hand corner. There is also more diffuse infiltration extending along the line of the hair follicles to just beneath the epidermis.

Horseshall and Essex 65

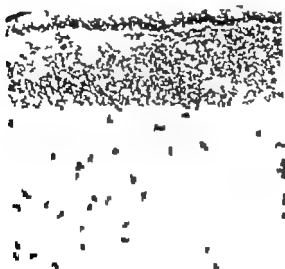


FIG. 199

Leprosy, cutaneous nodule.—The epidermis is stretched over an area of dermis which is heavily infiltrated by numerous inflammatory cells and granulomatous tissue.

Horseshall and Essex 80

LEPROSY

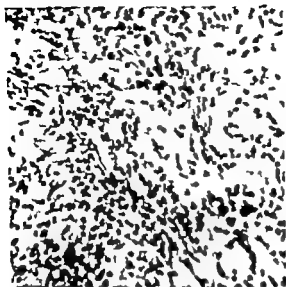


FIG. 200

Leprosy, high power, view of cutaneous nodule.—The infiltrate is seen to consist of great numbers of histiocytes and some fibroblasts forming matrix in which run capillaries.
Hermann and Essex, v. 275

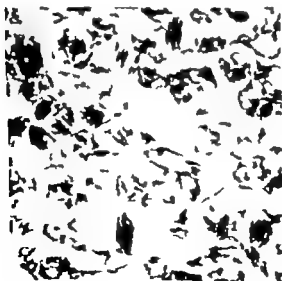


FIG. 201

Leprosy—T show the Lepros bacilli, stained red, some lying free, the majority packed within the large histiocytes ("Lepros cells")
Ziehl-Neelsen 1000

ACTINOMYCOSIS



FIG. 202

Actinomycosis—*Portion of wall of sinus*. There is an epithelial lining and the cavity of the sinus full of pus cells and compact clump of the streptothrix actinomycetes.
Hematoxylin and Eosin $\times 65$

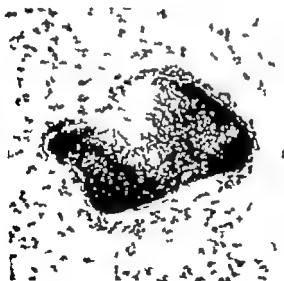


FIG. 203

Actinomycosis—*same as Fig. 202*—*Some of the contents from the sinus stained by Gram method, to show thick mass of the Gram positive filaments of streptothrix actinomycetes surrounded by pus cells.*
Gram Stain and Basic Fuchsin 275

CUTANEOUS LEISHMANIASIS



FIG. 204
Sand Fly—*Phlebotomus papatasi*
Shaw & Carter $\times 11$

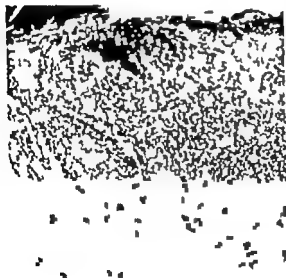


FIG. 205
Cutaneous Leishmaniasis (Oriental Sore) Early nodule.—The dermis is heavily infiltrated
by mononuclear cells of various types and the epidermis becoming acanthotic and
disrupted.
Hermann and Eason 100

CUTANEOUS LEISHMANIASIS



FIG. 206

Cutaneous Leishmaniasis (Oriental Sore) — *Edge of Sore* — The epidermis is ulcerated and the dermis is diffusely infiltrated by various types of mononuclear cells.

Henshaw and Evans $\times 75$



FIG. 207

Cutaneous Leishmaniasis (Oriental Sore) — Another portion which shows in addition to the diffuse cellular infiltration of the dermis, more localized nodules in which are low giant cells, the whole resembling tuberculous nodule.

Henshaw and Evans 55

CUTANEOUS LEISHMANIASIS

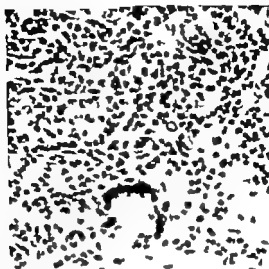


FIG. 208.

Cutaneous *Leishmaniasis* (Oriental Sore)—The same as Fig. 207—A more detailed view of nodule showing to be composed of histiocytes, some lymphocytes and giant cell
Hemadon and Evans $\times 375$



FIG. 209

FIG. 209—Cutaneous *Leishmaniasis* (Oriental Sore)—In this the Leishman-Donovan bodies are stained and are seen to be within the large histiocytes of the nodule
Leishman Stain 1,000



FIG. 210

FIG. 210—Cutaneous *Leishmaniasis* (Oriental Sore)—A smear from sore as the above—One large mononuclear cell (histiocyte) seen with many Leishman-Donovan bodies in its cytoplasm. The binucleate character of these bodies indicated

Leishman Stain 1,000

NAEVUS

The scope of the term *naevus* is sometimes restricted to a variety of cutaneous tumour in which the predominating cell is the pigmented or potentially pigment-producing type known as the *naevus cell*, but as a number of other tumours which occur in the skin have certain features in common with this particular example, although differing from it histologically it has been thought more appropriate to give the term a wider meaning and to define it as follows — A *naevus* is a circumscribed hyperplasia or tumour formation which has its origin in aberrant embryological cutaneous structures or "rests." *Naevi* may be congenital or may appear at any age and are of almost universal occurrence. They develop slowly and sooner or later with few exceptions, reach a stationary stage. In rare cases they undergo malignant changes.

As *naevi* may develop from any one of the elements which are normal constituents of the skin, and as in some cases several types of cells or structures are involved, the *naevi* present great variations in their histological appearance. They may be classified as follows —

DERIVED FROM THE EPIDERMIS.	<div style="display: inline-block; vertical-align: middle; font-size: 3em; line-height: 1;">{</div> <div style="display: inline-block; vertical-align: middle;"> <p>NAEVUS KERATOSUS.</p> <p>NAEVUS SENILIS.</p> <p>NAEVUS (EPITHELIOMA) CYLINDROMATOSUS (<i>Cylindroma</i>).</p> <p>NAEVUS PIGMENTOSUS.</p> </div>
DERIVED FROM THE EPIDERMAL APPENDAGES.	<div style="display: inline-block; vertical-align: middle; font-size: 3em; line-height: 1;">{</div> <div style="display: inline-block; vertical-align: middle;"> <p>NAEVUS PILOSUS, AND PILONIDAL SINUS.</p> <p>NAEVUS (EPITHELIOMA), ADENOIDES CYSTICUS.</p> <p>NAEVUS SEBACEUS.</p> <p>NAEVUS SEBOCYTOMATOSUS.</p> <p>NAEVUS SYRINGADENOMATOSUS (<i>Hydrocystoma</i>).</p> <p>NAEVUS SYRINGADENOMATOSUS PAPILLIFERUS.</p> </div>
DERIVED FROM COLLAGEN	<div style="display: inline-block; vertical-align: middle; font-size: 3em; line-height: 1;">{</div> <div style="display: inline-block; vertical-align: middle;"> <p>NAEVUS FIBROMATOSUS.</p> </div>
DERIVED FROM VASO-FORMATIVE CELLS.	<div style="display: inline-block; vertical-align: middle; font-size: 3em; line-height: 1;">{</div> <div style="display: inline-block; vertical-align: middle;"> <p>NAEVUS VASCULOSUS.</p> <p>LYMPHANGIOMA.</p> <p>HAEMANGIOMA.</p> <p>ANGIOKERATOMA.</p> <p>GLOMUS.</p> <p>ADENOMA SEBACEUM (PRINGLE).</p> </div>

NAEVUS KERATOSUS (Figs. 212 214). In this *naevus* the epidermis alone is involved, and the abnormality takes the form of an extreme degree of cornification. The thickened stratum corneum may simply form a compact mass which projects above the skin surface, or the epidermis may form a series of ridges between which the horny masses are embedded. The rete malpighii may be either acanthotic or diminished in thickness. The sweat and sebaceous glands in the area show no abnormality. There may be a small amount of lymphocytic perivascular infiltration.

These naevi usually have a linear distribution and are referred to clinically as *naevus linearis*, *naevus unius lateris*, and *ichthyosis hystrix*. The excessive cornification gives them a darkish colour but there is no increase in melanin, and no naevus cells (see later) are present. The developmental abnormality may be confined to the mouths of a few adjacent hair follicles, and the name *naevus acneiformis* has been given to this type.

In the absence of any knowledge of the etiology of the conditions known as *Darier's Disease*, *Porokeratosis*, and *Acanthosis Nigricans*, and as they present clinical features as regards development and course which bear some similarity to the naevi, they are included along with the hyperkeratotic naevi to which they bear some histological resemblance.

Darier's Disease (Figs. 215-217). The lesions of this rare condition, which presents a striking clinical picture, are brought about by a process of dyskeratosis, a premature and abnormal keratinisation of individual rete cells. The process of abnormal keratinisation affects individual cells, or groups of cells, and this change is apparent even in the middle rete and more prominently in the stratum granulosum. The cells in question lose their prickles, become detached from their neighbours so that they come to lie in a small lacuna, the cell membrane becomes thickened, the nucleus is pyknotic, the cytoplasm assumes a homogeneous hyaline appearance, and vacuolation may be seen. These dyskeratotic cells (*corps ronds*) are gradually pushed up into the stratum corneum which is itself thickened and tends to become invaginated as horny plugs into the subjacent epidermis. In this area of hyperkeratosis the dyskeratotic cells may still be discernible, or they may be represented only by fragments of their pyknotic nuclei (*grains*), the cell body having merged with the surrounding normal keratohyalin. There is a moderate degree of acanthosis, and a tendency to the downwards development of narrow and pointed epidermal processes. The epidermal proliferation never exceeds the bounds of an ordered hyperplasia, and malignancy has not been known to develop.

Porokeratosis (Fig. 218) consists of a localised area of hyperkeratosis, which usually involves the orifice of a sweat-gland duct, and which projects on the surface as a horny spur. These lesions are arranged in the form of a small circle, the original central area having become atrophic. The condition is often familial and hereditary and the lesions may assume a linear distribution.

Acanthosis Nigricans (Fig. 219) is a rare skin disease characterised histologically by hyperkeratosis and hyperpigmentation. As far as these features are concerned it bears a histopathological resemblance to *naevus unius lateris*. The disease may be naevoid in character—in some cases it is present at birth or appears early in life—and a familial tendency has been noted. It is therefore convenient to place it along with the true hyperkeratotic naevi, if only for comparison. The chief

interest in *acanthosis nigricans*, which is benign as far as its cutaneous manifestations are concerned, is that visceral carcinoma is associated with some fifty per cent of cases.

NAEVUS SENILIS (Figs. 220 and 221). This lesion is also known as *verruca seborrhoeica* and *verruca senilis*. It makes its appearance during or after middle age and consists of a flat brownish growth with an uneven fleshy surface which is usually covered with a pultaceous scale. It is frequently multiple, and occurs on the scalp, face and trunk. There is no evidence that it is allied to the contagious verrucae. On rare occasions it is the starting point of a squamous-cell carcinoma, but this occurs so seldom that it would be unjustifiable to place *naevus senilis* in the class of precancerous dermatoses.

Naevus senilis consists of a hyperplasia of the rete, which takes the form of a network of almost uniform thickness throughout the lesion. There is no tendency to the formation of downward projecting epidermal processes such as is seen in *verruca vulgaris*, nor is there vacuolation of the rete cells, or the development of eosinophil droplets within the cell cytoplasm. The stratum corneum is thickened and from its under surface horny masses project into the underlying rete. These cornified masses may branch so that on section isolated plugs of stratum corneum may appear to be imbedded in the substance of the rete. The hyperkeratosis gives a darkish colour to the clinical lesion, but there is no increase in the melanin content of the basal layer.

NAEVUS (EPITHELIOMA) CYLINDROMATOSUS (Fig. 222). This rare tumour also called *cylindroma*, is usually described along with basal cell carcinoma, although it shows no malignant tendencies of any kind. Its usual site is the scalp, where it occurs in varying numbers in an individual case, the lesions usually making their first appearance in childhood or adolescence. They vary in size from that of a pea to a tomato and tend to be somewhat pedunculated.

Histologically the tumour is composed of irregularly shaped masses of cells of basal type situated in the middle portion of the dermis. On section these collections are circular, oval, or bizarre-shaped. The cells are of two types, those at the periphery being smaller and more darkly staining than those in the centre. The cell masses are encircled by a band of homogeneous collagen, the same homogeneous material is present between the cells, and cavities of varying sizes are present in most of the cylindrical cell masses. The homogeneous enclosing collagen does not exhibit the staining properties of hyaline degeneration, and it is regarded as a primitive form of collagen.

NAEVUS PIGMENTOSUS (Figs. 223-250). This variety of *naevus* is characterised by the presence of pigment-forming cells in the dermis. These cells are referred to as *naevus cells*, they contain the pigment-forming oxydase and they are presumably of epidermal origin. They are quite distinct from the pigment-containing phagocytic wandering cells (*melanophores*) which are normally present in the dermis, and

which merely store and remove formed melanin but are incapable of producing it. The naevus cell is medium sized round oval, or polygonal in shape, and its nucleus is poor in chromatin. In the pigmented naevus these cells are present in varying numbers in the dermis. They are not distributed in any constant pattern, sometimes occurring as a dense mass, sometimes in columns set at right angles to the epidermis, and sometimes in small clumps. Their content of formed melanin is variable not infrequently they contain no melanin whatsoever and sometimes the basal cells of the epidermis are more highly pigmented than the subjacent naevus cells. Larger polygonal melanophores may be present amongst, or in the vicinity of the naevus cell masses. The epidermis overlying the naevus cells may be normal in appearance or it may be acanthotic and hyperkeratotic.

In the *Blue naevus* (Figs. 239-241) the naevus cells and melanophores are deeply placed in the region of the coils of the sweat glands.

Pigmented naevi may develop malignant tendencies and when this occurs the degree of malignancy is extreme. Histologically the malignant pigmented naevus, or malignant melanoma (Figs 244, 245 and 247-250) as it is usually called, shows dense penetrating masses of naevus cells, which are irregular in shape and size and which show numerous mitosis. There is a marked tendency for the malignant cells to penetrate the epidermis and appear in it as small clumps or nests. The decision as to whether a naevus is benign or malignant may present considerable difficulty in the early stages of the change from one state to the other (Figs 242 and 243), and in some cases both types of growth are to be seen in a single tumour.

Metastasis from a malignant melanoma takes place through the lymph channels and the blood stream, and the liver lungs and skin are frequent sites for the secondary growths. In the skin they frequently occur in showers, and spontaneous and complete regression of secondary skin growths has been noted. This appears to be brought about as a result of fatty degeneration of the tumour cells, the pigment being absorbed by histocytes.

NAEVUS PILOSUS (Fig. 251). In its simple form the naevus pilosus consists of a hyperplasia of hair follicles, mainly of the lanugo type. Surrounding the follicles there is a whorled agglomeration of collagen bundles and the intervening collagen is increased in amount and is more cellular than usual—in fact the mass may resemble a fibroma. The overlying epidermis is normal. These lesions usually occur on the face, and are particularly common in the region of the *alae nasi*, where they form rounded tumours, often with a slightly constricted base, which may reach half a centimetre or more in diameter.

Hair follicles in abnormally large numbers may be present in a pigmented naevus, a mixed variety of naevus being formed—*naevus pigmentosus et pilosus*.

The **PILONIDAL SINUS** (Figs. 252-254) arises in association with a localised hyperplasia of misplaced hair follicles—a developmental inclusion or rest—and it therefore falls into the category of naevus. The innumerable hair follicles are poorly developed and spring from the base and sides of a sinus which opens on to the surface. The upper portion of the sinus is lined with epidermal epithelium which is continuous with the adjacent surface epidermis. Infection of the sinus may take place with the production of an indolent subcutaneous abscess which tracks widely in the surrounding tissue and gives rise to multiple discharging sinuses. The pilonidal sinus generally occurs over the lower sacral and coccygeal regions.

NAEVUS (EPITHELIOMA) ADENOIDES CYSTICUM (Figs. 255 and 256). This structure was originally described as epithelioma adenoides cysticum, but it is a benign type of tumour which shows no epitheliomatous tendencies. It occurs clinically as multiple pin-head to pea-sized tumours situated on the face and trunk. It usually becomes apparent during adolescence.

Histologically it is composed of numerous cystic spaces containing keratin material, situated in the upper and middle areas of the dermis. The walls of these cysts have the structure of a hair follicle, and from the outer layer of cells on part of the circumference there arises a network of cellular strands. Similar strands may be seen projecting downwards from the basal layer of the overlying epidermis which is stretched and somewhat thinned. The cells composing these networks have the appearance of basal cells, but once the lesion has developed the cells show no further tendency to invade the surrounding dermis, and the junction between their strands and the surrounding fibrous tissue is clear cut. The supporting tissue is more cellular than normal but there is no inflammatory cellular reaction such as is usually seen in basal cell carcinoma.

NAEVUS SEBACEUS (Figs. 257-264). Clinically naevus sebaceus takes the form of a linear lesion on the scalp, the supra- or retro-auricular region, the forehead, the cheek, or the neck. The lesion is usually present at birth and is by no means an uncommon one. The surface of the tumour is flat and only slightly raised above the surrounding skin. The tumour is formed by a mass of firm yellowish papules, which are frequently umbilicated.

Histologically this naevus is composed of numerous hypertrophic sebaceous glands which are of normal structure. The ducts are dilated and are filled with sebaceous material and epidermal debris. There is no capsule enclosing the area, and the lesion is a hyperplasia of the sebaceous glands and not an adenoma. The sweat glands in the area may show cystic dilation of their ducts and of the coil portion. The epidermis on the area involved in the sebaceous gland hyperplasia may be thin and stretched in places, and in others show an acanthosis similar to

that seen in naevus senilis. A naevus sebaceus may in rare instances be the starting point for a carcinoma of either basal or squamous-cell type (Figs. 263 and 264).

NAEVUS SEBOCYSTOMATOSUS (Figs. 265 and 266). In some cases cystic dilation may affect large numbers of the sebaceous glands the condition being described as sebocystomatosis, or steatocystoma multiplex. The condition is often familial and should be considered as a naevoid type of cystic abnormality which does not result from obstruction to the sebaceous gland duct as in the case of the much more common sebaceous retention cyst. Apart from the absence of the plugged orifice the lesions of sebocystomatosis resemble those of the retention cyst and also milium lesions, although the latter are much more minute. In the example shown a cellular papilliferous process is present as an ingrowth from the cyst wall (naevus sebocystomatosus papilliferus).

NAEVUS SYRINGADENOMATOSUS (*Hydrocystoma*) (Figs. 267 and 268). The clinical appearance of this condition is that of a small somewhat translucent papule, and the lesions are usually multiple. The upper part of the trunk and face are the usual sites.

Histologically the tumour is composed of numerous dilated sweat gland ducts, which appear on section as cysts of varying sizes scattered profusely throughout the entire dermis.

There is a papilliferous variety—**NAEVUS SYRINGADENOMATOSUS PAPILLIFERUS** (Figs. 269-275)—which bears a close clinical resemblance to naevus sebaceus, and none to the simple syringadenomatous variety. It is usually found on the face, is often linear and in contrast to naevus sebaceus it not infrequently becomes eroded on the surface and presents a reddish moist appearance, resembling cruberant granulation tissue. The coiled secretory portion of the sweat duct may be normal in appearance or dilated. Above that level cystic dilations similar to those seen in naevus syringadenomatosis are to be seen and in the superficial zone of the dermis papillary growths project into the lumen of these cysts. Finally a dilated duct opens on to the surface through a wide cup shaped depression. This depression is lined with epithelium of sweat-gland duct type which is continuous at the periphery with the epidermis. Papillary processes rise from the floor of the depression and project towards the surface.

The cysts are lined by two layers of normal sweat-duct epithelial cells, and the papilliform projections consist of a fibrous tissue stroma covered by a similar layer of cells which is continuous with that lining the cyst. The fibrous tissue surrounding the cysts and composing the stroma of the papillary projections is densely packed with plasma cells. Some cysts are formed by dilatation of the coil portion of the sweat gland the epithelium of which is stretched and thinned.

In some cases there is a co-existing hyperplasia of sebaceous glands so that the lesion is a mixture of naevus sebaceus and naevus syringadenomatosis papilliferus.

NAEVUS KERATOSUS

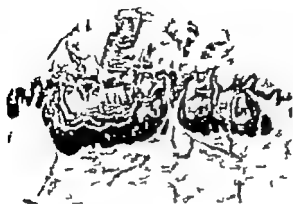


FIG. 212.

Nevus Unus Lateris—There is most marked degree of hyperkeratosis, the hypertrophied stratum corneum is as the area normal in structure but parakeratosis is present in places. The stratum granulosum is thickened. There is considerable degree of acanthosis, but this is regular in character. The papillae are hypertrophied, and there is small amount of perivascular lymphocytic infiltration. There are no nerve cells present.

Hematoxylin and Eosin $\times 18$

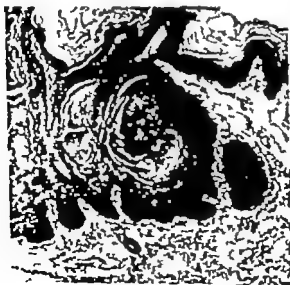


FIG. 213

Nevus Unus Lateris—This is higher magnification of Fig. 212 showing hyperkeratosis acanthosis and hypertrophy of the papillae.

Hematoxylin and Eosin $\times 90$



FIG. 214

Acute Ulcer Lesion—The hyperkeratosis does not protrude on to the surface as much as in Fig. 212. There are acanthosis, the rete being thinned and stretched and the pegs few and far between. Sweat-gland ducts are seen in the stratum corneum of the stratum. There is little or no perivascular lymphocytic infiltration. Sebaceous glands are present and are normal in appearance.

Hershen and Egan 50

DARIER'S DISEASE.



FIG. 215

Darier Disease.—A horny plug invaginates the epidermis and in it are to be seen small granules of degenerated nuclei. The epidermis is acanthotic, and groups of dyskeratotic cells is seen lying in clear space. There is some degree of lymphocytic infiltration in the subjacent dermis.

Hematoxylin and Eosin $\times 100$



FIG. 216

Darier Disease.—The horny plug contains keratinized dyskeratotic cells. One such cell is to be seen in the deeper part of the epidermis. There is acanthosis with the production of elongated downward-projecting columns of epidermis. In spite of this apparent tendency for the epidermis to invade the dermis, Darier disease does not develop malignant features.

Hematoxylin and Eosin 175.



FIG. 217

Darier. Disease—Dyskeratotic cells ("corps ronds") are to be seen just below the stratum granulosum. They are surrounded by clear space, and varying degrees of dyskeratotic changes are present. The cell membrane is thickened and vacuolation is present in one cell. In all of them the nucleus is pyknotic and the cytoplasm has hyaline appearance.

Herniman and Eason 300

POROKERATOSIS

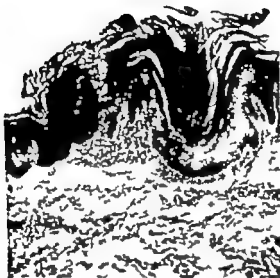


FIG. 218

Porokeratosis.—There is marked hyperkeratosis and some parakeratosis forming a heaped-up mass at the mouth of sweat-gland duct. The epidermis on both the upper and ventral is normal in structure, but owing to its being stretched the rete pegs are absent. There is some small round-cell infiltration in the dermis underneath the horny area.

Hamaker and Egan 70.

ACANTHOSIS NIGRICANS

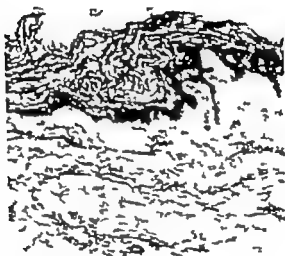


FIG. 219

Acanthosis Nigricans.—There is excessive cornification on the surface. The epidermis is atrophic and reduced to only one or two layers of epithelial cells and there is disappearance of the rete pegs. There is considerable amount of melanin pigment in the deepest layer of the epidermis, also some particles in the dermis. The dermis is dense and its collagen is hyaline.

Hamman, Egan 110



FIG. 220

Nevus Senilis. (Seborrheic Keratosis)—The main feature is irregular acanthosis, which forms network. There is some hyperkeratosis, and horny plugs project downwards into the rete. The basal layer is normal and there is no increase in melanin. The dermo-epidermic junction is well defined.

Hamman and Egan A 121



FIG. 221

Nevus Senilis. (Seborrheic Keratosis)—This shows an irregular retiform acanthosis, with no tendency to downward projection of the rete pegs. The intrusion of horny plugs from the surface into the rete is seen. The basal layer shows no accumulation of melanin granules, the frequent dark colour of the lesions being due to the hyperkeratosis. The dermo-epidermic junction is clearly demonstrated and there is the very slightest perivascular infiltration.

Hamman and Egan 65

NAEVUS CYLINDROMATOSUS



FIG. 222

Naevus Cylindromatosus — The tumour is composed of masses and cords of epithelial cells similar to those seen in the basal-cell carcinoma, which it resembles. The cells of the peripheral layer are cubical to columnar in form. Several of the epithelial masses show cystic spaces within them. There is well-defined supporting fibrous stroma. The epidermis is stretched out over the tumour but separated from it by zones of fibrous tissue.

Hermann and Egan $\times 65$.



FIG. 223

Nevus Pigmentosus (Melanoma).—There is much acanthosis of the epidermis and the dermal papillae are seen cut in various planes amongst the epithelium. These papillae are deposits of melanin pigment.

Hamaker and Essex $\times 12$



FIG. 224

Nevus Pigmentosus (Melanoma).—The same as Fig. 223, to show in more detail the pigment both in the dermal papillae and, to a lesser degree, in the basal cells of the epidermis. There is considerable lymphocytic infiltration around small vessels of the dermis.

Hamaker and Essex 65.

NAEVUS PIGMENTOSUS

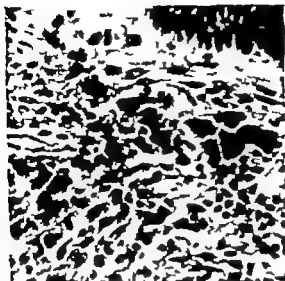


FIG. 23

Nevus Pigmentosus (Mole).—This is as Figs. 223 and 224 showing the melanocytes packed into large cells of histiocytic type (melanophores) lying in the meshes of dermal papillae. Around are scattered few non-pigmented nevus cells, some lymphocytes and fibroblasts. A small part of the basal layer of the epidermis shows no pigment in some cells.

Henshaw and Evans 400.



FIG. 26

Nevus Pigmentosus (Mole).—This is papillomatous structure thick covered by thin layer of stretched epidermis. The matrix extensively occupied by masses of nevus cells, amongst which are specks of pigment.

Henshaw and Evans 9

NAEVUS PIGMENTOSUS



FIG. 227

*Nevus Pigmentosus (Mole).—*A portion of the same series as Fig. 226, to show the epidermis and the numerous clusters of nevus cells occupying much of the connective tissue stroma.

Hematoxylin and Eosin $\times 90$

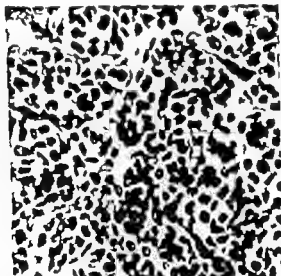


FIG. 228

*Nevus Pigmentosus (Mole).—*The same as Figs. 226 and 227. Here the characters of the nevus cells are well seen: they are polygonal cells with basally acicled cytoplasm and round or oval nuclei. Amongst them are several large, elongated histiocytes loaded with brown melanin pigment.

Hematoxylin and Eosin 450.

NAEVUS PIGMENTOSUS.



FIG. 229

Nevus Pigmentosus (Melanoma)—In this example the epidermis is stretched over the relatively deeply placed tumor, which consists of packed nevus cells and many pigmented cells. The tumor mass is well defined.
Hermann and Egan $\times 15$

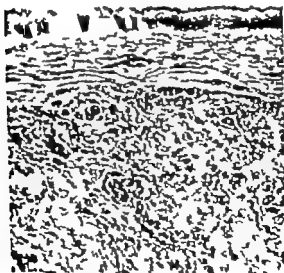


FIG. 230

Nevus Pigmentosus (Melanoma)—Edge of the same specimen as Fig. 229.—The densely packed nevus cells and interspersed pigment-bearing cells (melanophores) are shown in more detail. A small blood vessel seen at the side amongst the tumor cells.
Hermann and Egan $\times 100$

NAEVUS PIGMENTOSUS



FIG. 231

Nevus Pigmentosus of Palpebra (Melanoma) from chin—There is an excess of hair follicles with their associated sebaceous glands and some sweat glands. In the dermis between the hairs are small groups of nevus cells.
Hematoxylin and Eosin $\times 20$.

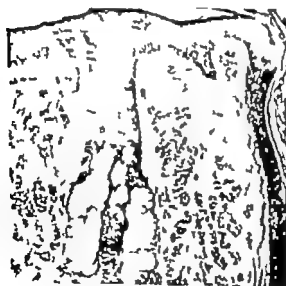


FIG. 232

Nevus Pigmentosus of Palpebra (Melanoma) from chin—A portion of the same nevus as Fig. 231 to show the groups of nevus cells in the dermis between the hair follicles.
Hematoxylin and Eosin $\times 105$.

NAEVUS PIGMENTOSUS

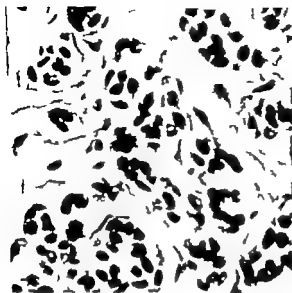


FIG. 233

Nevus Pigmentosus et Pilosus (Melanosis) from chin—Collection of typical nevus cells from the same specimen as Figs. 231 & 232

Hematoxylin and Eosin $\times 450$.



FIG. 234

Nevus Pigmentosus—Portion of nevus to show nevus cells in groups and singly in the dermis

Hematoxylin and Eosin 80

NAEVUS PIGMENTOSUS



FIG. 235

Nevus Pigmentosus (Mole)—This example is shown because of the amount and density of the stroma. In this stroma are groups of typical nerve cells, some brownish just under the epidermis—which is stretched over the surface. There are also groups of pigment-carrying cells (melanophores) in the superficial part.

Hermann and Egan $\times 60$.

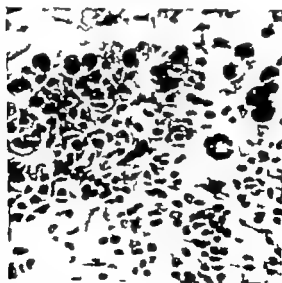


FIG. 236

Nevus Pigmentosus (Mole)—The nerve runs as in F. 235, superficial portion.—Along the edge of this lesion are many pigment-carrying cells (melanophores); the deeper cells are of ordinary nerve type.

Hermann and Egan 320

NAEVUS PIGMENTOSUS

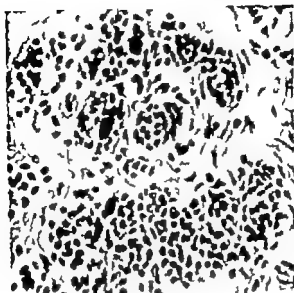


FIG. 237

Nevus Pigmentosus (Mole).—The same as Figs. 235 & 236, deeper portion, to show more nervous cells, fewer melanophores, and greater amount of fibrous strands.
Hematoxylin and Eosin. $\times 320$.

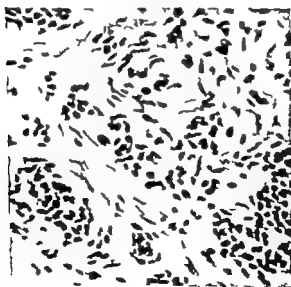


FIG. 238

Nevus Pigmentosus (Mole).—The same as Figs. 235, 236 & 237, deeper portion. Here the fibrous strands are abundant and many of the nervous cells are becoming isolated by it and are atrophic.
Hematoxylin and Eosin. $\times 320$.

NAEVUS PIGMENTOSUS



FIG. 39

Blue Nevus—The term "blue" refers to its clinical appearance. The section shows hyperkeratosis and slight hyperplasia of the epidermis, also some lymphocytic infiltration around the small vessels of the superficial dermis. The chief feature is the presence around the sweat glands of nevus cells and melanophores.

Henshaw and Egan $\times 90$.



FIG. 40

Blue Nevus. The same as Fig. 39, area of sweat glands.—The position of the pigment-containing cell will soon be seen here.

Henshaw and Egan $\times 75$.

NAEVUS PIGMENTOSUS



FIG. 241

Blue Nevus The same as Figs 239 & 240. A portion of sweat gland.—The cells bearing the melanin pigment lie in the stratum alongside the coil of sweat gland.

Hammann and Evans $\times 400$

NAEVUS PIGMENTOSUS.



FIG. 342

Naevus Pigmentosus (Melanoma).—This tumour illustrates the difficulty in deciding whether or not melanoma (pigmented naevus) is malignant. Parts were regular and benign, other parts had cells irregular in size, shape and arrangement. The portion portrayed shows much hyperkeratosis on the surface and very irregular and acanthotic epidermis. Lying in the situation of the dermal papilla are groups of cells of the ordinary nerve cell type, but mixed with these in places are large irregular cells carrying pigment. Along the lower edge of the epidermis are irregular pigmented cells.

Hamaker and Eason $\times 85$

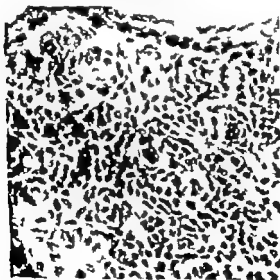


FIG. 43

Naevus Pigmentosus (Melanoma).—The same as Fig. 24, surface view. This illustrates the variety in size, shape and pigment-content of the tumour cells. The epidermis atrophic and stretched over the tumour. *Hamaker and Eason* 400

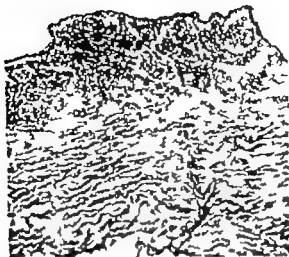


FIG. 244

Malignant Naevus Pigmentosus (Malignant Melanoma).—This tumour arose at one end of linear benign pigmented naevus (Fig. 246) which had been present for many years. This area is malignant. It consists of masses of tumour cells which are invading the epidermis and penetrating subcutis into the dermis.
Hemphill and Evans $\times 60$.

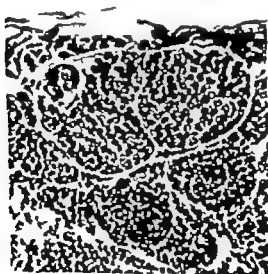


FIG. 245

Malignant Naevus Pigmentosus (Malignant Melanoma).—A portion of the surface of the preceding (Fig. 244) specimen to show the tumour masses invading the epidermis, which much enlarged. There is no definite evidence of any pigment in this portion.
Hemphill and Evans 330.



FIG. 246

Naevus Pigmentosus (Melanoma) non-malignant area — This specimen was taken from the opposite end of the pigmented part to that from which the malignant tumour (Fig. 244) arose. It mainly shows a number of large elongated cells heavily laden with dark brown (melanin) pigment.

Hematoxylin and Eosin $\times 200$

NAEVUS PIGMENTOSUS



FIG. 247

Malignant Nevus Pigmentosus (Malignant Melanoma)—A general view given of the tumor which consists of mass of nevus cells covered by epidermis stretched over the surface. It is all defined on its deeper aspect and few areas of pigment are seen.
Hermann and Euse 29

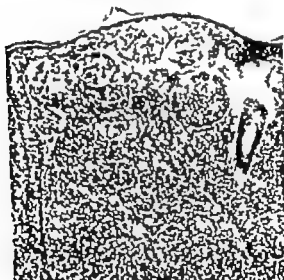


FIG. 248

Malignant Nevus Pigmentosus (Malignant Melanoma)—The same as Fig. 47 surface part of tumor.—The epidermis on the surface thinned and being recessed upon by irregular groups of large cells of which some show pigment granules. A little deeper are number of deeply pigmented cells, and few lymphocytes in the meshes of the connective tissue. Deeper still the tumor cells are more uniform in size, densely packed, and show only few pigmented cells, which are indistinguishable from nevus cells.

Hermann and Euse 110

NAEVUS PIGMENTOSUS



FIG. 249

Malignant Nevus Pigmentosus (Malignant Melanoma)—The same as Figs. 247 & 248, middle zone of tumor.—The tumor cells here are irregularly arranged in masses with scanty stroma in which run delicate blood vessels. The cells themselves are irregular in size and shape and some show pigment.

Henshaw and Essex 170



FIG. 250

Malignant Nevus Pigmentosus (Malignant Melanoma)—The same as Figs. 247, 248 & 249, deeper zone.—In this part very few tumor cells are present but many large histiocytes loaded with melanin pigment are seen. The stroma is more abundant and very vascular.

Henshaw and Essex 170

NAEVUS PILOSUS

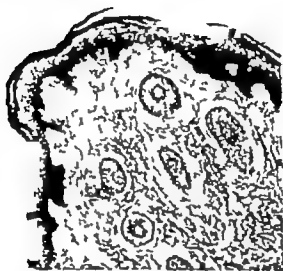


FIG. 251

Nævus pilosus—The main feature of this specimen is the relatively large number of hairs—cut on cross section. The stroma fibrous and there are no nevus cells.
Hemmelin and Egan 80

PILONIDAL SINUS



FIG. 252

Nerve Pilonidal (Pilonidal Sinus)—The surface epithelium extends as lining into the sinus but stops abruptly before the base is reached. Surrounding the whole area of the sinus is thick zone of dense chronic inflammatory fibrous tissue.

Henshaw and Egan × 45



FIG. 253

Nerve Pilonidal (Pilonidal Sinus)—Middle zone, where the epithelial lining terminates and the sinus beyond is lined by inflammatory fibrous tissue, in which are hair follicles. The sinuses are many small hairs cut in various planes and mixed with epithelial debris.

Henshaw and Egan 14

PILONIDAL SINUS

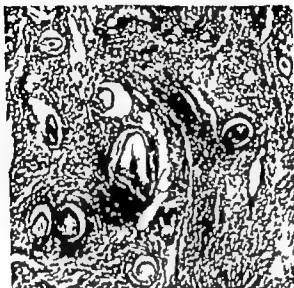


FIG. 284

Acute Pilonidal (Pilonidal Sinus) part of wall of deeper area — Numerous hairs, mostly of small size, are cut transversely or obliquely and are surrounded by fibrous tissue in which are small blood vessels and many chronic inflammatory cells.

Henshaw and Evans, x 85

NAEVUS (EPITHELIOMA) ADENOIDES CYSTICUM.

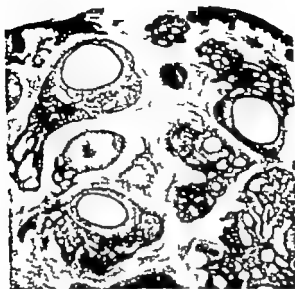


FIG. 255

Naevus (Epithelioma) Adenoides Cysticum.—There is proliferation of hair follicles, from the outer surface of back, and from the basal layer of the epidermis, anastomosing strands of basal cells radiate into the surrounding dermis; cystic spaces are forming in the centre of epithelial masses. The stroma which supports this cellular network is rich in connective tissue cells.
Hematoxylin and Eosin $\times 25$.

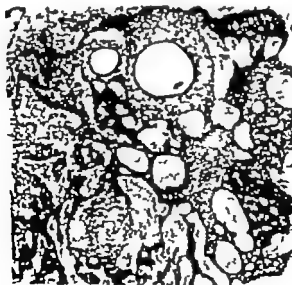


FIG. 256

Naevus (Epithelioma) Adenoides Cysticum.—High power view of Fig. 255.
Hematoxylin and Eosin $\times 100$

NAEVUS SEBACEUS.



FIG. 257

Naevus Sebaceus—Low power view showing hyperkeratosis, acanthosis, proliferation of sebaceous glands, cystic dilation of sweat-gland ducts, no cellular reaction and no nevus cells.

Hematoxylin and Eosin $\times 10$.



FIG. 258

Naevus Sebaceus—Higher power view of Fig 257 showing hypertrophy and proliferation of sebaceous glands, and some lymphocytic infiltration. Nevus cells are absent.

Hematoxylin and Eosin 25.

NAEVUS SEBACEUS

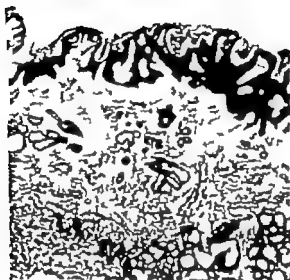


FIG. 259

Nævus Sebaceus.—Higher power view of Fig. 257 showing hyperplastic, anastomosing, occasional cystic dilatation of sweat-gland ducts, and some dilatation in the end of the sweat-gland (bottom right).

Hamaker and Egan 25

NAEVUS SEBACEUS.



FIG. 260

Naevus Sebaceus.—The tumour composed of masses of hyperplastic sebaceous glands situated immediately beneath the epidermis, which is raised up over them.
Hermann and Egan $\times 20$



FIG. 261

Naevus Sebaceus. The same as Fig. 260.—T shows in more detail the sebaceous glands, one of which opening on the surface. There is some lymphoid infiltration between the glands and the epidermis but no nerve cells are present.
Hermann and Egan 73

NAEVUS SEBACEUS

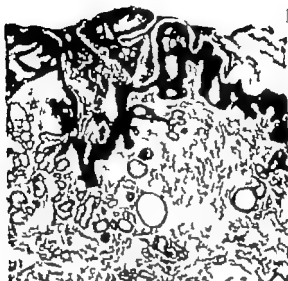


FIG. 262

Nævus Sebaceus.—Portion of *naevus sebaceus* showing hyperkeratosis, plugging of sebaceous gland orifices, regular acanthosis, hypertrophy and proliferation of sebaceous glands to the left of the section, cyst formation in sweat-gland ducts, and very slight degree of lymphocytic infiltration. There are no malignant changes present in this area.

Hamman and Egan 20.



FIG. 263

Nævus Sebaceus showing malignant change.—*Acantholytic epidermis* from another portion of the same *naevus sebaceus* as shown in Fig. 262. This section shows small encysting nodule of basal cell carcinoma at the left-hand side of the section.

Hamman and Egan 65.

NAEVUS SEBACEUS

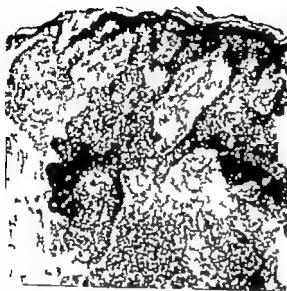


FIG. 264

Nevus Sebaceus showing malignant change—Acute cystic degeneration from some sebaceous elements as shown in Figs 262 and 263. A squamous cell carcinoma is developing, and there is an intense surrounding lymphocytic and polymorph infiltration.

Hersules and Egan 65

NAEVUS SEBOCYSTOMATOSUS



FIG. 265

Naevus Sebocystomatosus, Papilliferous type. Portion of cystic area—The wall of the cyst is lined by thin layer of cells but in places papillary invaginations arise from these and project into the cyst. One small papilla is seen attached to the wall and part of larger one lying within the cyst. The wall of the cyst is formed by layers of dense fibrous tissue. *Hammann and Egan 15.*



FIG. 266

Naevus Sebocystomatosus, Papilliferous type. The small papilla shown in preceding figure (Fig. 265)—The single layer of cells forming the lining of the cyst is seen and it is continued into the papilla which is composed of smaller cells closely packed. Amongst them are polar areas which are delicate processes of the supporting stroma carried in with the epithelium as cover to the epithelial groups of cells forming the papilla. The role is the very early formation of another projection, seen as small aggregation of the epithelial cells around portion of stroma. The underlying stroma is formed of dense fibrous tissue. *Hammann and Egan 95.*

NAEVUS SYRINGADENOMATOSUS



FIG. 267

Nevus Syringadenomatosus.—Cystic dilatation of the sweat gland ducts constitutes the lesion. The ducts are more numerous than normal. In this specimen there was no evidence of hyperplasia of the sebaceous glands and no papilliferous process were found projecting into the cysts.

Hamaker and Egan $\times 40$.

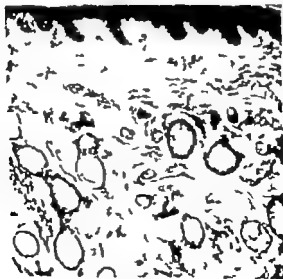


FIG. 268

Nevus Syringadenomatosus.—Throughout the middle and lower portions of the dermis there are numerous dilated ducts of sweat glands.

Hamaker and Egan 100

NAEVUS SYRINGADENOMATOSUS PAPILLIFERUS



FIG. 269

Naevus Syringadenomatosus Papilliferus.—Cystic dilatation of varying degree is seen in the sweat-gland ducts superficial to the coil. Ducts are seen to open on to the surface through large cup-shaped apertures. The epithelial cells lining this aperture are similar to those of normal sweat-gland duct, and are continuous at the sides with the epidermis. From the floor of the cup-shaped depression numerous papillary processes project towards the surface. They consist of fibrous tissue stroma covered by sweat-gland duct epithelium. The stroma is packed with plasma cells.

Hamman and Egan $\times 8$



FIG. 270

Naevus Syringadenomatosus Papilliferus.—This shows papilliferous processes projecting on to the surface from floor of sweat gland duct epithelium. The epidermis has disappeared. The sweat-gland ducts are tortuous and show varying degree of dilatation. Plasma cells are numerous in the stroma of the processes.

Hamman and Egan 22

NAEVUS SYRINGADENOMATOSUS PAPILLIFERUS

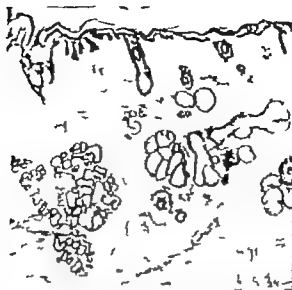


FIG. 271

Nevus Syringadenomatosus Papilliferus.—Low power of field adjacent to that shown in Fig. 270, at the edge of the tumor. The epidermis is intact here, there is tortuosity and dilatation of the sweat-gland ducts. The sebaceous glands are normal. There are areas showing compact infiltration of plasma cells.

Hamman and Evans $\times 22$.



FIG. 272

Nevus Syringadenomatosus Papilliferus.—A higher power view of Fig. 271 to show normal end-epithelium and cystic dilatation of the more superficial ducts.

Hamman and Evans $\times 80$.

NAEVUS SYRINGADENOMATOSUS PAPILLIFERUS



FIG. 273

Naevus Syringadenomatosus Papilliferus.—A cystic dilation of sweat gland duct opening on to the surface. The adjacent epidermis is continuous with the wall of the cyst. Papilliferous processes project into the interior of the cystic opening.
Hematoxylin and Eosin $\times 30$.



FIG. 274

Naevus Syringadenomatosus Papilliferus.—Surface cystic dilation similar to that shown in Fig. 273. The papilliferous projections are covered with sweat-gland duct epithelium, and the interior contains numerous plasma cells.
Hematoxylin and Eosin $\times 50$.

NAEVUS SYRINGADENOMATOSUS PAPILLIFERUS



FIG. 275

Naevus Syringadenomatosus Papilliferus.—This section shows from above downwards, the stems of papilliferous process packed with plasma cells, the lower epithelium of sweat gland duct forming covering for the process, the lumen of the cavity and its coverings of similar epithelium and some plasma cells in the adjacent dermis.

Hamaker and Essex 450



FIG. 276

Naevus fibromatosus.—Thin pedunculated papillous covering of matrix of loose connective tissue in which are thin-walled blood vessels and covering which is layer of epidermis showing reticular acanthotic arrangement and some hyperkeratosis. The stalk is covered with stretched epidermis.

Hemadine and Emsw 14

VASCULAR NAEVI

LYMPHANGIOMA (Fig. 277) is composed of dilated lymph spaces of varying size and shape. These cavernous spaces may occur at any depth in the dermis, but when situated immediately beneath the epidermis they have a clinical appearance of small tense bullae resembling frog spawn, to which the term *lymphangioma circumscriptum* (Fig. 278) is applied.

The **HAEMANGIOMA** is a much more cellular tumour than the lymphangioma, and three distinct varieties occur

The *capillary angioma* (Figs. 279-284) consists of packed masses of angioblastic cells and capillaries formed by them, situated in the upper dermis.

The *cavernous angioma* (Fig. 285) occupies a deeper situation in the dermis and in addition to angioblastic cells and aberrant capillaries, large irregularly shaped thin-walled blood spaces are present.

Angioma serpiginosum (Figs. 286 and 287).—This is merely a variant of the capillary angioma. It extends irregularly throughout the dermis and even more deeply the strands of vaso-formative cells and vessels may be considerably separated in places by collagen fibres. Oedema and infiltration by inflammatory cells may be superadded but are not essential features of the condition.

ANGIOKERATOMA (Figs. 288 and 289) This condition occurs on the fingers, toes, knees, elbows, and scrotum and generally establishes itself at an early age. In a typical case there is also a history of acrocyanosis or erythema pernio.

The histological picture shows marked hyperkeratosis, acanthosis and hypertrophy of the rete pegs, so that the rete on section is in the form of a network. In the papillae and superficial part of the dermis, there are dilated blood vessels and lacunae containing blood, a varying amount of polymorph infiltration, and some fragmentation and disintegration of collagen fibres and elastic tissue.

GLOMUS TUMOUR (Figs. 290-292). This is a specialised form of vascular growth which has the characters of the normal glomus—a mechanism for regulating the blood supply in certain parts of the body especially the fingers and toes. The tumour consists of vascular channels with an endothelial lining, immediately outside of which are several layers of polygonal cells (the "glomus cells") Around some of the vessels there is a supporting collagenous framework which contains plain muscle cells and abundant nerve fibres, mainly of the non-myelinated type. The nature of the glomus cell is debateable, it is regarded by some as an angioblast, even capable of forming elastic fibrils, by others it is

considered to be a myoblast. Because of the rich nerve supply these tumours are usually painful.

ADENOMA SEBACEUM (Figs 293 and 294). This tumour occurs as a small red nodule varying in size from a pin head to a pea. It is situated on the face, is always multiple, and is found in mental defectives.

The title adenoma sebaceum is a misnomer for the histological appearance is that of an angioma. The sebaceous glands are certainly large and moderately numerous, but this is only to be expected in the skin of the nose and cheeks, and in association with a liberal vascular supply.

The epidermis is stretched and thinned and the dermis contains numerous blood spaces and capillaries in varying degrees of dilatation. The supporting connective tissue is more cellular than normal, and the sebaceous glands are large but show no pathological changes and are not abnormally numerous. The lesions, unlike those of naevus sebaceus, do not tend to become malignant.

Malignant vascular tumours—Angioendotheliomata—of the skin are rare. They are rapidly growing tumours composed of primitive vessels and angioblastic cells infiltrating into the surrounding tissues. The **KAPOSI TUMOUR** (Figs 295-298), may be regarded as of this nature. In it there is a formation of many irregular vascular spaces and channels, which frequently rupture and give rise to hæmorrhages. Between these there are many undifferentiated mesoblastic and inflammatory cells. The tumour tends to infiltrate both the epidermis and the deeper tissues and it may regress and recur.

LYMPHANGIOMA.

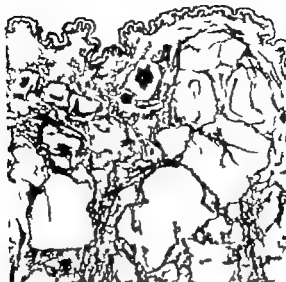


FIG. 277

Lymphangioma.—This tumor consists of large, thin-walled cavernous lymph spaces filled with coagulated lymph. These spaces extend from just under the epidermis through the whole thickness of the dermis. The epidermis has been pushed upwards by the mass of spaces. The supporting connective tissue is stained blue.

Hutchinson Ann. Surg. & B.

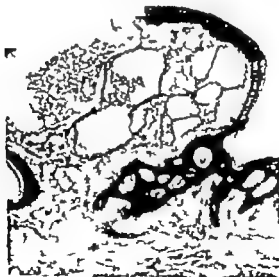


FIG. 278

Lymphangioma (circumscription type).—This form of lymphangioma also consists of large cavernous spaces lined by flattened endothelial cells—not always discernible—and delicate reticular supporting tissue. The lymph content has been fixed in situ in several spaces. This type of lymphangioma always situated superficially in the papillary area of the dermis and the spaces abut on the epidermis.

Hutchinson and Ewart 95



FIG. 279

Capillary haemangioma (vascular naevus—strawberry type).—This shows the whole extent of the tumour which stretches from the base of the epidermis through the dermis and into the subcutaneous tissue. Hair follicles are seen to be surrounded by it, and in the subcutaneous tissue, fat spaces and also a few large normal blood vessels are seen in its substance.

Hemadon and Eason $\times 6$



FIG. 280

Capillary haemangioma (vascular naevus—strawberry type).—A higher power view of its superficial portion. The tumour tissue consists of closely packed capillary vessels, with few larger blood spaces, and extends practically to the epidermis, occupying the dermal papillae. Part of hair follicle seen at the bottom right.

Hemadon and Eason 55.

HAEMANGIOMA.

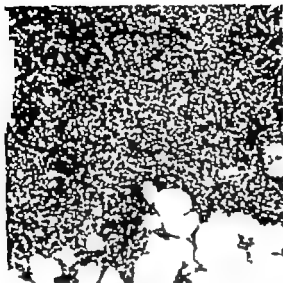


FIG. 281

Capillary haemangioma (vascular sarcoma—strawberry type)—High power view of the deeper portion. Part of the subcutaneous fat is seen with tumour abutting on it. The tumour here is composed of closely packed endothelial cells amongst which only few minute capillary lumina have developed.

Hamshon and Evans $\times 145$

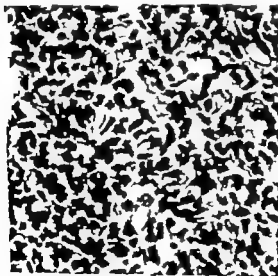


FIG. 282

Capillary haemangioma (vascular sarcoma—strawberry type)—High power view of the preceding showing the endothelial type of cell and few developed capillaries.

Hamshon and Evans $\times 475$.

HAEMANGIOMA.



FIG. 283

Capillary haemangioma ("vascular naevus") showing disposition in the dermis similar to those of Fig. 279, but having larger blood spaces.
Henshaw and Evans $\times 14$



FIG. 284

Capillary haemangioma ("vascular naevus")—A higher power view of the surface. There is the same endothelial neo-formative tissue as seen in Fig. 280 but in this case it is developing into larger blood spaces, almost approaching the cavernous type.
Henshaw and Evans 75.

HAEMANGIOMA.



FIG. 285

Cavernous haemangioma—1. The blood spaces are large and thin-walled. There are also areas with small capillary-type vessels and endothelial cells.

Hamman and Egan 20



FIG. 286

Angioma serpiginosum.—This variant of the hemangioma. It has the same two-layered cells and blood spaces formed from those which vary in size from capillary to vesicle. The tumor is ill-defined, being irregularly distributed in the cutaneous and subcutaneous areas.

Hamaker and Egan $\times 110$.

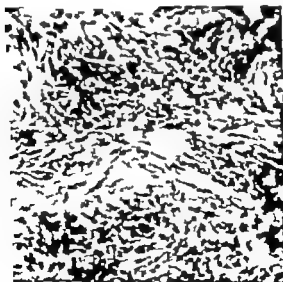


FIG. 287

Angioma serpiginosum.—T show in more detail number of small capillary spaces and their attendant cells. These are embedded in fibrous stroma.

Hamaker and Egan 210



FIG. 288

Angiokeratoma.—This shows hyperkeratosis, acanthosis and papillary hypertrophy; blood spaces and dilated vessels—some perivascular leucocytic infiltration; fragmentation of collagen fibres in the middle dermis.

Henshaw and Essex 60.



FIG. 289

Angiokeratoma.—The features are similar to those shown in Fig. 288. Note that the lacunae which are situated within the area of the epidermis are actually bordered by wall of fibrous tissue.

Henshaw and Essex 30.

GLOMUS TUMOUR.



FIG. 290

Glomus tumour.—This consists of cavernous blood spaces, the walls of which are thicker than those of the preceding haemangiomas.

Henshaw and Egan $\times 20$.

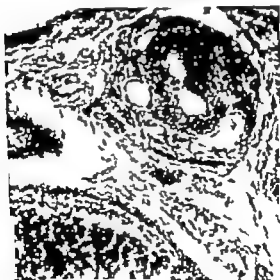


FIG. 291

Glomus tumour.—Higher power showing the edge of large blood space and several small spaces. The spaces have an internal lining of endothelium surrounding which is made of several layers of glomus cells. The nature and function of these cells are the subject of conjecture. They have been regarded as being of endothelial origin and also as primitive plain muscle cells (myoblasts). Nerve fibrils—not stained in this preparation—are usually numerous around the vascular spaces.

Henshaw and Egan 110.

GLOMUS TUMOUR.

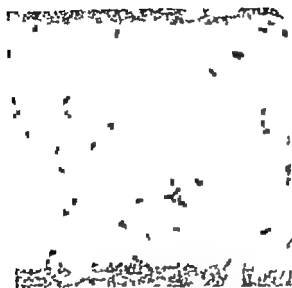


FIG. 292

Glomus tumour.—This example shows the greater part of the tumour to be composed of the glomeruli. Although these are blood spaces, many small, few large. There is delicate reticular stroma between the groups of cells.

Picro-haematoxylin $\times 100$

ADENOMA SEBACEUM.



FIG. 293

Adenoma Sebaceum.—This shows hyperplasia of the sebaceous glands, and dilated blood spaces.
Hammon and Eason $\times 15$

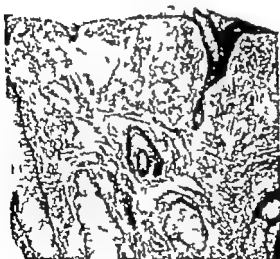


FIG. 294

Adenoma Sebaceum.—A high power view of Fig. 293 to show anastomosing blood spaces of varying sizes. The tumour is combination of anaplastic tissue and glandular hyperplasia.
Hammon and Eason $\times 70$.

KAPOSI SARCOMA.

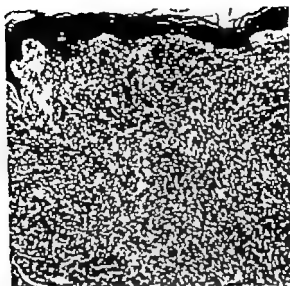


FIG. 295

Kaposi Sarcoma—A form of *Angio-sarcoma*; skin area. The epidermis is stretched and thin with slight hyperkeratosis. The tumour occupies the dermis and extends deeply to the underlying tissues. This view shows the numerous small cells with surrounding supporting cells and haemorrhage into the stroma.

Harrison and Egan $\times 75$

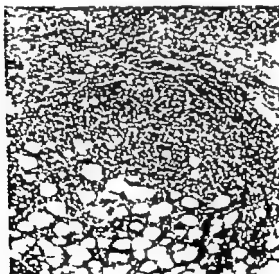


FIG. 296

Kaposi Sarcoma—The same case as Fig. 295, subcutaneous area. The fatty tissue is infiltrated in the same fashion as the skin.

Harrison and Egan $\times 75$.

KAPOSI SARCOMA.



FIG. 297

Kaposi Sarcoma—The same case as Figs. 295 & 296 showing the invasion of muscle by the tumor.

Hemphill and Enns $\times 75$.

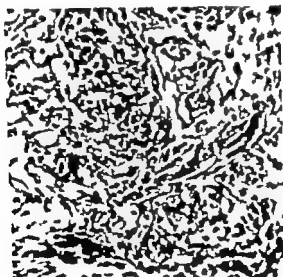


FIG. 298

Kaposi Sarcoma—The same case as Figs. 295, 296 & 297. A detailed view in which can be seen several small vessels accompanied by spindle-shaped fibroblastic cells cut in various planes.

Hemphill and Enns 275

CYSTS

Cutaneous cysts can be placed in the following categories —

CONGENITAL

NAEVUS SYRINGADENOMATOSUS

NAEVUS SYRINGADENOMATOSUS PAPILLIFERUS.

NAEVUS SEBOCYSTOMATOSUS

EPITHELIOMA (NAEVUS) ADENOIDES CYSTITICUM.

PILOIDAL CYST

ACQUIRED

SEBACEOUS RETENTION CYST (Figs. 299-302)

MILIUM (Figs. 303-305).

IMPLANTATION CYST (Figs. 306-308).

MUCOUS (OR MYXOMATOUS) CYST (Figs. 309 and 310)

CYSTIC BASAL-CELL CARCINOMA (Figs. 344-346).

The congenital cystic tumours fall into the category of *naevus* and are described in that general section, while the cystic variety of basal-cell carcinoma is illustrated and described in the section on carcinoma.

Of the acquired cysts, the sebaceous cyst and milium are of common occurrence, the implantation cyst is not infrequently seen, but the mucous cyst is of extreme rarity. The sebaceous retention cyst is caused by prolonged blockage of a sebaceous gland orifice by a comedo, or by a superficial infective process producing complete stenosis of the duct. The result is a dilatation of the gland with atrophy of all but the periphery of the lobules, and the formation of a sac filled with keratin and sebaceous material. The wall of the cyst is composed of a number of epithelial cells arranged in layers resembling a stretched portion of epidermis. The contents of the cyst may become calcified.

Milium occurs on the forehead, the temples, the eyelids and the malar regions and cheeks. The lesions may be single or multiple and are sometimes numerous. The cyst is small and is situated immediately under the epidermis, being separated from it only by a narrow strip of dermis. The cyst wall is formed by two or three layers of flattened epidermal cells and the contents are strands of keratin material. These small cysts often extrude themselves from the skin spontaneously the pressure which they exert causing an atrophy of the overlying structures. No reliable evidence is available to indicate their mode of origin, but it is presumed that they are derived from lanugo hair follicles, or perhaps from sweat-gland ducts.

The implantation cyst is produced by the mechanical intrusion of a portion of the epidermis into the dermis as a result of trauma. The

wall is formed by epithelial cells arranged in layers corresponding to those of the epidermis, and direct continuity between the epidermis and the wall of the cyst may be traced in some cases. The contents of the cyst are keratin strands.

The mucous cyst occurs in the region of the finger nails, usually on the dorsum of the terminal phalanx or on the side of a finger. It is produced by a mucinous degeneration of collagen fibres in a localised area of the dermis. There is no epithelial lining to the cyst the wall of which is formed by collagen fibres. In places the fibres immediately surrounding the cystic space are thin and form an open lace work in which mucin can be demonstrated but the major portion of the wall of the cyst is composed of collagen fibres which are normal in appearance. The mucous cyst is difficult to eradicate, and recurrence may take place even after the apparent complete excision of the affected area.

SEBACEOUS CYST

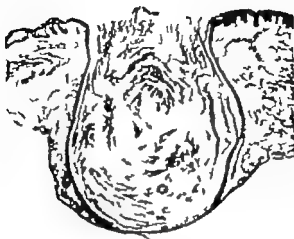


FIG. 299

Sebaceous Retention Cyst—The entire sebaceous gland, filled with keratin and sebaceous material, as glandular structure is lost and reduced to one, the wall of which consists of few flattened epithelial cells. The exterior of the follicle is greatly dilated, and the epithelial wall of the cyst is continuous with the epidermis.

Henshaw and Egan $\times 20$.

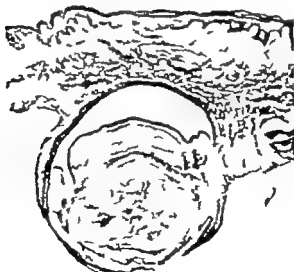


FIG. 300

Sebaceous Retention Cyst—This section is on a different plane from that in Fig. 299 and shows the keratin-containing one completely shut off from the surface.

Henshaw and Egan $\times 20$.

SEBACEOUS CYST



FIG. 301

Sebaceous Cyst—The glandular structure of the sebaceous gland has disappeared, and the gland has become transformed into an epithelial-lined sac containing keratin.

Harrison and Evans 23



FIG. 302

Upper of Sebaceous Cyst—In the upper portion is seen the keratinized contents of the cyst immediately beneath this thin layer of epidermal epithelium bounded by the fibrous tissue of the dermis.

Harrison and Evans 140

MILIUM.

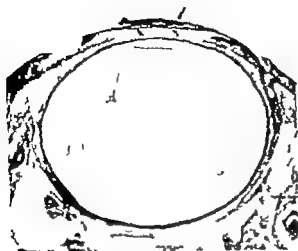


FIG. 303

Milium.—The cyst situated in the superficial portion of the dermis, and separated (from the stretched epidermis) by thin band of dense tissue. Serial sections give no indication of the tissue from which the cyst has been derived, nor does it open on surface.

Hammon and Egan $\times 25$

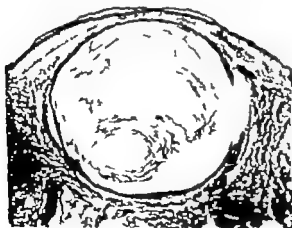


FIG. 304

Milium.—The contents of the cyst are composed of strands of keratin.

Hockenham, Allen, Stam $\times 25$

IMPLANTATION CYST

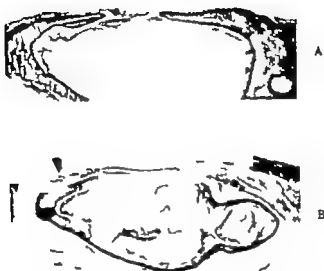


FIG. 306

Implantation Cyst—A—Shows the point at which the epidermis became invaginated to form the cyst, there being continuity of the cyst wall and the overlying epidermis. B—The cyst cut in different plane so that it appears unconnected with the surface. The wall of the cyst is formed by a thin layer of flattened epidermal cells which are undergoing extensive cornification and the contents of the cyst is a quantity of shed cornified material.

Harrison and Egan 10

IMPLANTATION CYST



FIG. 307

Implantation Cyst—The contents are strands of
keratinized material
Hemphill and Evans 18



FIG. 308

Implantation Cyst—The wall several cells thick, and is identical
in modified and compressed epidermal structure. Stratum
granulosum cells are present and fully developed keratin has
been produced

Hemphill and Evans 470,

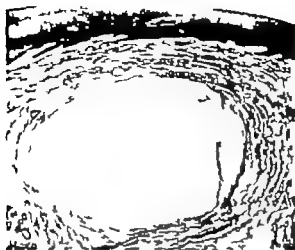


FIG. 309

Mucous Cyst—There is no cellular lining to the cyst, which occurs as a cavity in the dermis resulting from arrested degeneration of collagen.
Henshaw and Evans $\times 30$



FIG. 310

Wall of Mucous Cyst—This is composed of dense collagen fibres, such as some places are needed to form a fine open network showing traces of mucous.
Henshaw and Evans $\times 110$.

TUMOURS

Tumour formation may develop in any of the epidermal structures or the component parts of the dermis, so that both benign and malignant growths which are commonly seen in other tissues may also be met with in the skin. Tumours such as molluscum contagiosum, the contagious verrucae, and certain of the naevi are peculiar to the skin alone.

THE FIBROMA (Figs. 311 and 312) is to be distinguished from an ordinary nodule of scar tissue, or a bunion due to irritation and pressure, by the whorled arrangement of the fibrous strands composing it. It is usually well defined and dense, and numerous fibroblasts may be present or it may be composed almost entirely of collagen fibres, depending on its age and rate of growth. The cellular form may recur on removal and ultimately develop definite malignancy as a fibro-sarcoma.

MOLLUSCUM FIBROSUM (Fig. 313) is a multiple form of fibroma. The nodules may be pedunculated and the matrix oedematous or even myxomatous. While the tumours are usually distributed along cutaneous nerves there are frequently no nerve elements demonstrable in the superficial nodules.

FIBROUS EPULIS (Fig. 314) is yet another variety of fibroma, found along the gum or tooth margin.

CHONDROMA (Figs. 315 and 316). This is an unusual form of cutaneous growth, but it may form as a "callosity" e.g. over the knuckles of the fingers. It has the usual characters seen in chondromata in other situations—hyaline cartilage bounded by fibrous tissue.

MYOMA (Figs. 317 and 318), of the plain muscle variety—Leiomyoma—occurs in the dermis. It is a rare tumour and the usual site is the face. It is small in size and is composed of whorls of plain muscle fibres.

XANTHELASMA, XANTHOMA (Figs. 319-322) AND XANTHOMA TUBEROSUM MULTIPLEX (Figs. 323-325) must be considered as doubtful tumours from the pathological standpoint, although clinically they can be placed in the tumour group. In all three varieties there is a localised hyperplasia of histiocytes in the upper and middle dermis. These cells appear large, clear and vacuolated (foamy-cells) in ordinary paraffin sections, but in frozen sections appropriately stained they are seen to be laden with fat or lipid. In xanthoma tuberosum numerous large multinucleated histiocytes (Touton's giant-cells) are also present.

A local intra-cellular or a general metabolic upset in lipid metabolism is the immediate cause, and xanthoma and xanthoma tuberosum occur in association with diabetes (compare with necrobiosis lipoidica). Xanthelasma is found on the eyelids in otherwise healthy individuals of all ages.

In all three types of xanthoma the cells involved belong to the reticulo-endothelial system and the hyperplasia is of a type which permits of their inclusion along with the reticuloses.

THE CONTAGIOUS VERRUCAE (Figs. 326-336).

This group of contagious cutaneous papillomas comprises several well defined varieties, each of which is a modification of a uniform type of epidermal and dermal hypertrophy. The various representatives of the group have the following clinical titles — *Verruca Plana*, *Verruca Vulgaris*, with its subdivisions, *Filiformis*, *Digitata*, *Plantaris*, and *Condyroma Acuminatum*.

In *verruca plana* the hyperplasia is limited to the epidermis. This tissue is uniformly thickened and shows a hyperkeratotic stratum corneum in which parakeratotic cells may occur in small groups, an increase in the thickness of the stratum granulosum which varies directly with the thickness of the overlying horny layer and marked acanthosis which may have the appearance on section of a thick meshed network in its deeper part owing to inclusion of the papillae. The basal layer is regular and normal in appearance.

In *verruca vulgaris* there is a similar hyperplasia of the epidermis, but a special feature is the elongation of the rete pegs, which project downwards as finger like processes showing a tendency to curve inwards towards the central axis of the lesion. There is a corresponding elongation of the papillary processes. The stratum corneum is greatly thickened and in it are to be found layers of parakeratotic cells. The cells of the basal layer are normal, and the dermo-epidermal junction is regular in outline. Sweat-gland ducts may traverse the lesion and in some cases there is a mild degree of inflammatory reaction. In the *digitate* and *filiform* varieties the hyperplastic formation projects upwards from the surface of the skin as a group of narrow finger-like processes, while in the *verruca plantaris* the whole lesion is compressed into the substance of the dermis and is surrounded by a callosity. In the *condyroma acuminatum* the whole process has a more solid and succulent appearance than in the other varieties.

Degeneration of the rete cells may be seen in all varieties of verrucae and it bears no relation to the age of the lesion. It takes the form of vacuolation of the cell and the development of eosinophil droplets, which may be kerato-hyalin in the cell cytoplasm. The nucleus is pyknotic and pushed to one side, the cell membrane is thickened and the prickles are lost. This process may affect single cells or groups of cells, it occurs at any point in the rete above the basal layer and the degenerated cells eventually appear in the stratum corneum. The degenerative change reaches its most extreme degree in the apparent complete liquefaction of the cell which is transformed into an eosinophil staining anuclear mass. A number of adjacent cells may undergo this complete dissolution, and partially degenerated cells may be found

embedded in such a homogeneous mass. Cellular degeneration is seen most constantly and in its most advanced stages, in the verruca plantaris. That it is a process peculiar to the verrucous tissue and not dependent merely on pressure or to the heaping up of stratum corneum is shown by the fact that it does not affect the epidermis which forms the callosity surrounding the verruca, the rete and stratum corneum of this surrounding hypertrophied epidermis being sharply demarcated from the adjacent degenerated rete and stratum corneum of the verruca (Fig. 333). The degenerative change may ultimately involve the entire verruca with the result that it separates and is extruded (Fig. 334), thus bringing about a spontaneous cure of the condition.

The verrucae are differentiated from the senile keratoma by the degree of acanthosis and the regularity of the basal layer which they present, and by the constant presence of an inflammatory reaction at the base of the keratoma.

In the senile or seborrhoeic wart (Figs. 220 and 221) the acanthosis invariably presents the appearance of a network on section, horny plugs project downwards from the surface into the epidermis, and the lesion has a flat appearance, features which serve to distinguish it from the contagious verrucae. In the condyloma acuminatum there is a verrucous epidermal hyperplasia which is absent in the condyloma latum (Fig. 182) and in the latter there is the dense plasma cell infiltration common to all syphilitic lesions.

MOLLUSCUM CONTAGIOSUM (Figs. 337-339).

This lesion consists of an epidermal hyperplasia which is caused by the action of a filterable virus on the cells of the rete. A small tumour varying in size from a pinhead to a pea is produced in this way.

The hyperplasia of the epidermis is considerable and it projects into the dermis forming a mass with a lobulated outline. At the same time it protrudes above the level of the skin surface and carries a covering of the surrounding stretched epidermis with it. The junction between the basal layer of the epidermal mass and the dermis is regular and the cells of the basal layer appear normal. In the lower strata of the prickle celled layer eosinophilic inclusion bodies appear in individual cells and as the superficial central portion is approached an increasing number of cells contain these bodies. The stratum granulosum is thickened and the cells containing the inclusion bodies pass through it and form a dense mass superficial to it, where they are surrounded by a small amount of keratin. These eosinophilic inclusion bodies are pear shaped, and their narrow pole is more translucent than the rest of the body giving the appearance of a vacuole. They lie in a cavity in the cell protoplasm, and the cell nucleus is pushed to one side. By appropriate methods they can be removed from the containing cell and shown to consist of an envelope which encloses numerous elementary bodies.

BASAL-CELL CARCINOMA (Figs. 340-346) This tumour is of slow growth and is only locally malignant. It is composed of masses of small, round or polygonal deeply staining epithelial cells of basal-cell type. The cells which bound the masses at their periphery may be more columnar and as a result of compression all the cells may become spindle-shaped. Degeneration and liquefaction of groups of cells may produce cystic spaces of varying size within the tumour masses.

The invading cells penetrate the dermis for varying depths, and in old-standing tumours they may reach the subcutaneous tissue, muscle cartilage, and bone. The masses may take the form of solid columns or of thin strands which form a network. The point of origin may be either the epidermis itself or the epithelium of the hair follicles, sebaceous glands, sweat-glands, and sweat ducts. A tumour formation which arises from the epidermal appendages has no continuity with the epidermis itself. The cell masses, whatever their point of origin, are always clearly demarcated from the surrounding dermis which shows a variable degree of fibroblastic reaction and an infiltration with lymphocytes. Superficial ulceration of the central portion takes place sooner or later.

In a proportion of basal-cell carcinomas there is an admixture of tumour growth of squamous-cell type (Figs. 363 and 364) and such mixed forms are to be regarded as squamous-cell tumours from the point of view of malignancy.

SQUAMOUS-CELL CARCINOMA (EPITHELIOMA) (Figs. 347-362). This form of epidermal carcinoma grows rapidly, is both locally destructive, and gives rise to metastases. The epithelial down-growths take the form of groups and masses of cells of a type resembling those of the rete mucosum, with or without prickles. In the slower growing varieties the tumour cells tend to become differentiated in the same way as those of the rete mucosum with the resultant formation of cornified areas ("pearls" or cell-nests). In the rapidly penetrating anaplastic forms the tumour cells tend to be elongated or spindle shaped and they show no tendency in cell-nest formation. The tumour almost always originates from the epidermis rather than from its appendages, and the infiltrating cell masses are vague in outline and show no clearly defined margin as in the case in the basal-cell variety. Around all forms at the growing margin there is an inflammatory reaction ranging from slight to dense leucocytic and lymphocytic infiltration, and the formation of fibroblastic and fibrous tissue.

Chronic slow growing varieties do sometimes occur and in them the invading processes are often blunted and seem as if their growth had been arrested by the reaction in the surrounding tissues. Degeneration may occur in the cells in the centre of the processes and this may be followed by the deposition of calcium (Fig. 358).

A tendency to extreme cornification is a feature of squamous-cell carcinomas which develop on skin which has been subjected to long

continued irritation from paraffin and X-rays. The development of carcinoma in *Lupus vulgaris* is almost invariably due to the large and repeated doses of X-rays to which cases of this disease were subjected in the past.

A frequent precursor to squamous-cell carcinoma is a small localised area of hyperkeratosis, which reaches its most extreme degree in the CUTANEOUS HORN (Fig. 365).

PAGET'S DISEASE, OR INTRA EPIDERMAL CARCINOMA (Fig. 366). This condition is restricted to the skin of the breast, particularly that of the nipple, and is regarded as an invasion and permeation of the epidermis by tumour cells derived from the lacteal ducts. The malignant cells arising therein grow along the ducts and on reaching the epidermis they permeate its substance. Here they appear as groups of large clear cells lying in lacunae amongst the epidermal cells and producing a thickening of the epidermis while at the same time destroying its solid cohesive structure, interfering with the normal process of keratinisation, and leading to parakeratosis and superficial ulceration.

Another malignant condition which shows a clinical resemblance to Paget's disease, but which differs from it histologically is PSORIASIFORM CARCINOMA (Figs. 367-370). This variant of cutaneous carcinoma is characterised clinically by its resemblance to a plaque of psoriasis, by the slow peripheral extension of its thread-like edge, and by the tendency to atrophy and spontaneous cure of the older central areas. It may remain superficial throughout its course, showing no tendency to deep penetration but in a proportion of cases this does ultimately occur. The term includes Queyrat's erythroplasia and Bowen's pre-cancerous dermatosis. Histologically at the edge of the lesion, there is a basal-cell type of overgrowth which encroaches on the papillary layer of the dermis, and invades the epidermis. In the more central areas the rete pegs are lost, the basal-cell overgrowth has disappeared, and there is fibrosis and some lymphocytic infiltration in the papillary layer.

Such plaques are frequently multiple, and it sometimes happens that one of the lesions, or a portion of a large plaque, may assume a much higher degree of malignancy than the remainder. This may take the form of a more rapidly growing type of basal-cell tumour or the character of the growth may change to that of a squamous-cell carcinoma.



FIG. 311

Fibroma.—The tumor is sharply defined from the surrounding tissues which it compresses to form capsule around it, and it is covered closely by the epidermis which is stretched over it and shows hyperkeratosis. The tumor is compact, the fibrous tissue staining uniformly pink.

From Hamman and Van Gieson, p. 15



FIG. 312

Fibroma.—Higher power to show the characters of the tumor. It is composed of banded strands of collagen (stained blue) running in many places. In the center of some bands there are small, well formed blood vessels. There are few fibroblasts in this specimen.

Henderson Allen Shaw 70



FIG. 313

Molluscum fibrosum.—One of the many superficial nodules. The epidermis is thickened and stretched over the underlying nodules. The contents of cellular fibrous tissue fairly well defined from the deeper dermis and showing number of small vessels. No nerve fibres are present.

Hammon and Egan 60



FIG. 314

Fibrous epul.—This shows the same features as *Glossoma*.
Iron, Hammon and Van Gieson 25

FIBRO-CHONDROMA.



FIG. 315

Fibro-chondroma, from the back of the proximal interphalangeal joint of finger—This an unusual form of tumour. The section shows it to have dense fibrous periphery and more homogeneous cartilaginous centre. There is hyperkeratinosis on the surface. The tumour may have begun as fibrous growth and undergone metaplasia to cartilage in the central part. There is no obvious capsule to be seen.

Hemphill and Egan 20

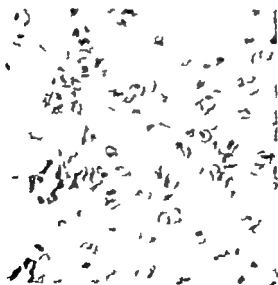


FIG. 316

The same—High power to show the homogeneous matrix and cells of the cartilage type.

Hemphill and Egan 250

LEIO-MYOMA.



FIG. 317

Leio-myoma.—The tumor is composed of bundles of muscle fibres and its position and general outline are evident. It lies in the dermis, slightly separated from the epidermis which is stretched over it, and in its deeper aspect, the muscle bundles are interspersed by fibrous strands.

Hamman and Essex 15



FIG. 318

Leio-myoma.—A higher magnification of Fig. 317 to show the bundles of plant vessels interspersed by pink-stained strands of fibrous tissue.

From Hamman and Essex 15.

XANTHOMA.



FIG. 319

FIG. 319 *Xanthoma*.—A portion of the nodule to show the surface epidermis, and the dermis extensively occupied by large, pale-staining cells.
Hematoxylin and Eosin $\times 75$



FIG. 320

FIG. 320 *Xanthoma*.—This is the view given by staining, from a section for fat—which is still present in the tumor and takes on the yellow dye.
Sudan III and Hematoxylin $\times 30$.



FIG. 321

FIG. 321 *Xanthoma*.—A detail view of the preceding (Fig. 319), illustrating the vacuolated cytoplasm of the large cells of the nodule.
Hematoxylin and Eosin 350

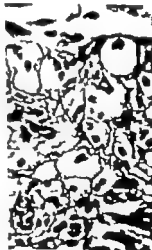


FIG. 322

FIG. 322 *Xanthoma*.—The same as Figs. 319-321, but better displayed by the staining method employed.
Hematoxylin Azan Stain 350

XANTHOMA TUBEROSUM.



FIG. 323

Xanthoma Tuberosum.—A—This is the superficial half of the tumour nodule and on its surface is the stretched epidermis. The nodule consists of great collection of cells of basocystic type infiltrating the dermis. amongst them are number of giant cells.
B—Deeper half of the nodule. The same picture is presented as in A, but the deep margin is clear cut and bounded by fibrous tissue of the dermis.

Harrison and Essex 15.

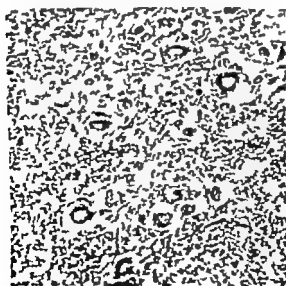


FIG. 34

Xanthoma Tuberosum.—The same as Fig. 323 to show large giant cells (Tendon giant cells) lying amongst the pale-stained connective tissue of the nodule.

Harrison and Essex 110

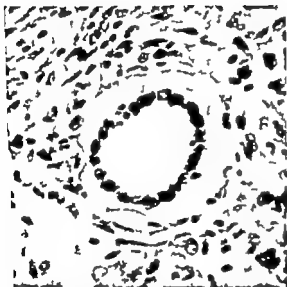


FIG. 325

Xanthoma Tuberosum, seen at Figs 323 & 324—A cyst cell (Tubercle giant-cell) with zone of vacuolated appearance due to lipid dissolved out in the preparation of the section. The surrounding cells have slightly vacuolated appearance from the same cause.

Henshaw and Egan 450

VERRUCA PLANA



FIG. 326

Verruca Plana—The section shows acanthosis, and hyperkeratosis.
Hammann and Egan $\times 25$.

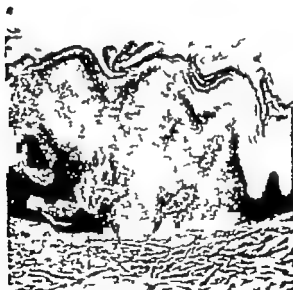


FIG. 327

Verruca Plana—There is hyperkeratosis and some parakeratosis. The rete is
 and the stratum granulosum shows an uneven hyperplasia. A change of
 is present in the region of the stratum granulosum, and below it.
Hammann and Egan 65.

VERRUCA VULGARIS



FIG. 328

Verruca Vulgaris showing hypertrophy of all the layers of the epidermis, complete absence of degenerative changes in the cells of the rete, finger-like processes projecting downwards from the rete and curving upwards towards the central axis of the lesion. There is an absence of any inflammatory reaction.

Hamman and Egan $\times 10$.



FIG. 329

Verruca Vulgaris—This lesion of long standing, which has taken the form of cauliflower surface excrescence.

Hamman and Egan $\times 10$.

VERRUCA PLANTARIS



FIG. 331

Verruca Plantaris.—This lesion shows all the features of verruca vulgaris, modified owing to constant external pressure. A most marked degree of osteoplastic degeneration is seen in many of the rete cells.
Hamman and Egan 12

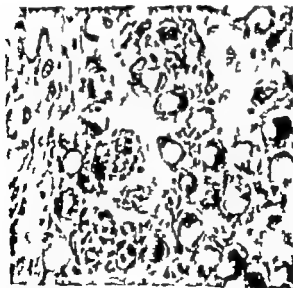


FIG. 332

Verruca Plantaris.—This shows clumps of degenerating cells embedded in a keratinous structure formed by the complete dissolution of surrounding cells. The degenerating cells contain large vacuoles and eosinophilic droplets. The cell membrane is thickened, and the prickles have disappeared.
Hamman and Egan $\times 475$.

VERRUCA PLANTARIS.



FIG. 333

Verruca Plantaris and surrounding cellular structure.—The structure of the surrounding cellular tissue is that of regular and orderly hypertrophy of all the epidermal layers, and no degenerative process is evident. It is clearly and sharply demarcated from the verrucal structure which has undergone gross cellular degeneration, evident both in its rate and stroma connective

Hematoxylin and Eosin $\times 45$.



FIG. 334

Verruca Plantaris.—The cells of the lower have undergone complete transformation into homogeneous eosinophil staining mass, as result of which spontaneous cure has been brought about.

Hematoxylin and Eosin $\times 55$

CONDYLOMA ACUMINATUM.



FIG. 335

Condyloma Acuminatum.—This lesion from the labium shows the usual changes of verruca vulgaris. Cellular degeneration present, and in the dermis there is no inflammatory reaction, but some vascular dilation. The lesion has more succulent appearance than that seen in verruca situated on less moist areas.

Henshaw and Essex 20



FIG. 336

Condyloma Acuminatum.—There are areas of hyperkeratosis, and degenerated rete cells are included in the hyperplastic stratum corneum. Groups of rete cells are vacuolated and contain eosinophil droplets.

Henshaw and Essex 75

MOLLUSCUM CONTAGIOSUM.



FIG. 337

Molluscum Contagiosum.—A general view of small tumor showing the lobulated masses of hyperplastic epidermis protruding into the dermis, and producing tumor which projects above the level of the surrounding skin surface, carrying covering of normal epidermis with it. Inclusion bodies are seen in increasing numbers from the lower rete to the central superficial area overlying the stratum granulosum.

Hamaker and Essex $\times 20$



FIG. 338

Molluscum Contagiosum.—The eosinophilic inclusion bodies are first seen in small numbers in the lower portion of the rete. They become increasingly numerous as the stratum granulosum is approached, and the central depression on the surface of the lesion.

67rd alk keratinized cells containing inclusion bodies

Hamaker and Essex 70

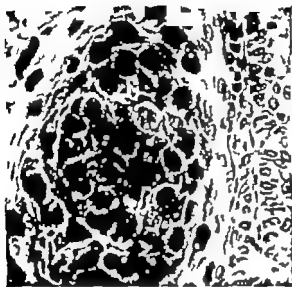


FIG. 339

Molluscum Contagiosum.—The eosinophilic inclusion bodies are pyriform or oval in shape, and many of them appear to contain small nuclei at their narrow end. This is actually an area where the containing membrane is thinner than over the rest of the body. The nucleus of the epithelial cell is pushed to one side.

Herman and Egan 400.



FIG. 340

Basal-Cell Carcinoma.—This specimen was from a young female, age 18. There is marked hyperkeratosis. At the extreme margins are portions of normal epidermis but in the central area this has been largely replaced by masses of deeply-staining cells of basal-cell type which also penetrate into the subjacent dermis. The downward projecting masses of basal-cells take the form of columns which are clearly defined from the surrounding stroma.

Hematoxylin and Eosin $\times 15$



FIG. 341

Basal-Cell Carcinoma.—This shows in more detail the epithelial processes composed of relatively small, closely packed and deeply staining cells resembling the basal cells of the epidermis. Between them is an overgrowth of cellular fibrous tissue in which are blood vessels.

Hematoxylin and Eosin 75

BASAL-CELL CARCINOMA.



FIG 342

Basal-Cell Carcinoma—This specimen shows much larger tumor mass composed of many thick columns. They are well defined at their edges and within them are scattered small cystic spaces.
Hematoxylin and Eosin $\times 18$

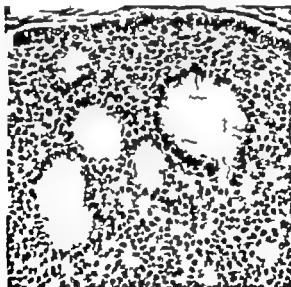


FIG 343

Basal-Cell Carcinoma. The same as Fig 342—Part of tumor mass showing the uniform character of the cells. At the invading edge the cells are more columnar in shape. In the substance are several small cystic spaces lined by columnar cells and containing coagulated albuminous fluid.
Hematoxylin and Eosin 200.



FIG. 344

Basal-Cell Carcinoma (Cystic type)—The epidermis stretched and stratified over the tumour which consists of two masses of basal cells. The centre of each mass is represented by large cystic spaces.

Henshaw and Essex $\times 11$

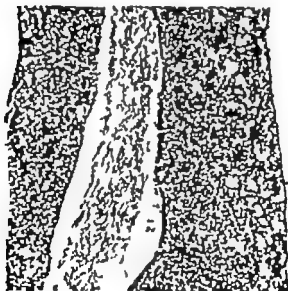


FIG. 345

Basal-Cell Carcinoma (Cystic type) The same as Fig. 344—A portion taken from the adjacent parts of the two nodules. In the centre is a strand of fibrous stroma. On either side are the masses of epithelium amongst which are little spaces due to degeneration of tumour cells. The edges of each cystic space shown in the low power (Fig. 344) appears at the margins of the illustration—it is formed by a layer of flattened, atrophic tumour cells.

Henshaw and Essex 110

SQUAMOUS-CELL CARCINOMA.



FIG. 347

Squamous Cell Carcinoma (Epithelioma).—From the lip. At the surface is a small portion of unaffected epidermis which changes into the tumour formation by an abrupt dipping down into the dermis. Further over are many more irregular processes cut in various places and in their central parts can be seen cornified areas, or 'cell-nests'. In the dermis surrounding the tumour masses there is heavy infiltration by chronic inflammatory cells of mononuclear type.
Hamaker and East. 40

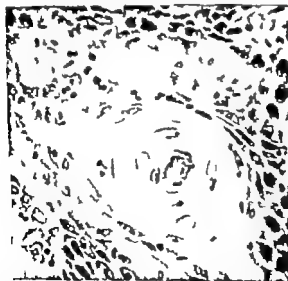


FIG. 348

*Squamous Cell Carcinoma (Epithelioma). The same as Fig. 347—*a cell-nest in the centre, a border of epithelial cells undergoing keratinization, around them are flattened squamous cells, and at the periphery are typical cells of rete mucosum type with intercellular bridges. A few inflammatory cells lie amongst the tumour cells, they are easily identified by their smaller size and darkly staining nuclei.
Hamaker and East. 400

SQUAMOUS-CELL CARCINOMA.



FIG. 349

Squamous-Cell Carcinoma (Epithelioma)—An early tumour with the downgrowths of epithelial processes still superficial and well-defined, but with typical central cornification ("cell-nest") in them.

Hematoxylin and Eosin $\times 45$.

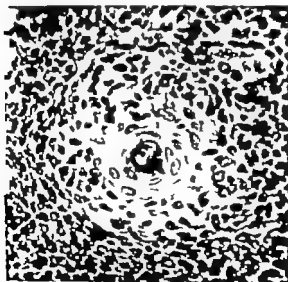


FIG. 350

Squamous-Cell Carcinoma (Epithelioma)—This tumour arose from the sebaceous. A typical cell nest from one of the epithelial processes is shown.

Hematoxylin and Eosin 400

SQUAMOUS-CELL CARCINOMA



FIG. 351

Squamous Cell Carcinoma (Epithelioma)—In this example there is invasion of the dermis by abundant tumour tissue but central keratinization is imperfect in there. There is heavy infiltration of the stroma by chronic inflammatory cells.

Hematoxylin and Eosin $\times 70$

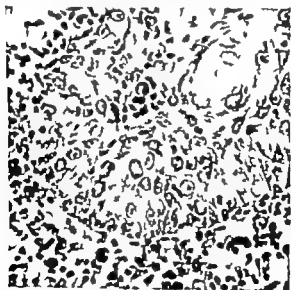


FIG. 352

Squamous Cell Carcinoma (Epithelioma) The same as Fig 351. A portion of tumour present shows as a substance small cell-nest surrounded by squamous epithelial cells. In the adjacent stroma are many chronic inflammatory cells—lymphocytes, plasma cells, and larger histiocytes.

Hematoxylin and Eosin $\times 400$



FIG. 353

Squamous-Cell Carcinoma (Epithelioma) - Anaplastic type. Only portion of an epithelial process is shown but it is typical of the others. There is no differentiation with formation of "cell-nests," the tumour cells being so closely packed that their squamous characteristics are difficult to determine. Many cells have anastotic figures in them which stain darker and are more compact than the ordinary nuclei.

Hematoxylin and Eosin $\times 175$

SQUAMOUS-CELL CARCINOMA.



FIG. 354

Squamous-Cell Carcinoma (Epithelioma).—This specimen was taken from the lip of man, aged 64, who had tumor there for three years. This is portion of more recent actively growing area. There is extensive surface cornification and thick layers of tumor tissue, most of which have extensive central degeneration with actual cornification in few places.

Hematoxylin and Eosin $\times 20$

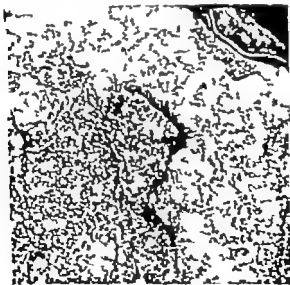


FIG. 355

Squamous-Cell Carcinoma (Epithelioma).—This shows in more detail the swelling and degeneration of the cells in the center of the tumor process with an imperfect cell-nest at the side of one process.

Hematoxylin and Eosin 80.

SQUAMOUS-CELL CARCINOMA



FIG. 356

Squamous-Cell Carcinoma (Epithelioma)—From the same patient as the specimen shown in Fig. 354. This part of the tumour has been present for three years. It is relatively benign epithelioma showing extensive degeneration in the central parts of the broad processes.

Hamaker and Egan 20



FIG. 357

Squamous-Cell Carcinoma (Epithelioma)—To show in more detail portion of the tumour shown in Fig. 356. In the center is large area of debris formed by broken down tumour cells, outside of this is wide zone of poorly staining clear cells undergoing degeneration. At the periphery are the ordinary squamous cells incident into the area of degeneration on the one side and sharply defined on the other side by the fibrous stroma.

A portion of the dermis included beyond that.

Hamaker and Egan 80.



FIG. 358

Cervical Squamous-Cell Carcinoma (Epithelioma).—This was a very slow growing tumour with extensive necrosis. The epithelial processes are short and many of cervical debris in which are dark blue-staining deposits of calcium.

Harrison and Egan p. 75

SQUAMOUS-CELL CARCINOMA.



FIG. 359

Squamous-Cell Carcinoma. Paraffin Carcinoma.—The epidermis hyperbolic. In the subjacent dermis there is widespread infiltration by the epithelial processes of the tumor. Cells show central cornification in the superficial area but little differentiation deeper at the growing margin.

Hematoxylin and Eosin. $\times 125$

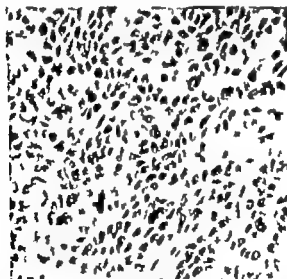


FIG. 360

Squamous-Cell Carcinoma. Paraffin Carcinoma.—Deep part of the tumor shown in Fig. 359. The epithelial processes are numerous, ill-defined, and composed of polygonal cells with no attempt at differentiation, an anaplastic type of squamous-cell carcinoma (epithelioma).

Hematoxylin and Eosin. $\times 300$

SQUAMOUS-CELL CARCINOMA.



FIG. 361

X-ray Carcinoma—There is much surface keratinization and typical down-growth of epithelial processes, many of which have the usual central keratinization. It is squamous-cell carcinoma (epithelioma).

Hamaker and Essex $\times 20$.



FIG. 362

Squamous Cell Carcinoma intermingled upon Layer I of epidermis—No trace remains in the section of the original lupus, but the dermis is extensively infiltrated by an epithelioma which has undergone extensive cornification.

Hamaker and Essex 25

CUTANEOUS HORN



FIG. 365

Cutaneous Horn—A thick, hyperplastic layer of epidermis forms the base from which projects a pillar of hard keratinized material. This horn is fissured on the exterior owing to its hard and tough character. Amongst the horny scuties are blue-stained calcareous deposits. The unaffected epidermis of normal thickness seen at the edges of the hyperplastic base.

Hammon and Egan 7



FIG. 366

Intra-epidermal Carcinoma—Paget Disease—The epidermal epithelium is invaded and largely replaced by the "Paget cells"—large, pale-staining clear cells. In the corresponding dermis there is great inflammatory reaction in the form of granulation tissue heavily infiltrated with inflammatory cells.

Harrison and Egan 65.

PSORIASIFORM CARCINOMA SHOWING DIFFERENT
TYPES OF MALIGNANCY IN VARIOUS AREAS
OF THE SAME LESION



FIG. 367

Psoriasiform carcinoma.—Here are seen downgrowths of the epidermis with the formation of processes and masses of epithelial cells, having the arrangement and characters of an epidermoid carcinoma of basal-cell type. *Hematoxylin and Eosin* $\times 100$.



FIG. 368

Psoriasiform carcinoma.—In this area there is much hyperplasia of the epidermis, the cells of which are large and clear. The deeper layers are commencing to penetrate into the sub-
jacent dermis and show an early attempt at central cornification. Here the malignancy is of the squamous epithelioma type. *Hematoxylin and Eosin* 70.

PSORIASIFORM CARCINOMA SHOWING DIFFERENT
TYPES OF MALIGNANCY IN VARIOUS AREAS
OF THE SAME LESION



FIG. 369

Psoriasiform carcinoma—This area shows only hyperplasia of the epidermis, with some parakeratosis superficially and excess of nuclei in its deeper layers. There is lymphocytic infiltration in the dermis. *Hermann and Eason* 110



FIG. 370

Psoriasiform carcinoma—There is marked parakeratosis. The rete pegs are hyperplastic, and a small area in the center shows early squamoidity. *Hermann and Eason* 100

INDEX

The black type indicates the principal reference

	Page		Page
Acanthosis ..	2	Capillary angioma	377 381 383, 385
nigrosus	494, 389	Carcinoma—analaplastic squamous—	
Acne vulgaris	187	cell	489
comedo stage	193	basal cell ..	455, 457
pustule	193	cystic type	450 461
Actinomyces	377	basal squamous	451, 461
Actinomycetes	33, 283	intra epidermal	453, 465
Adenoma sebaceum	378, 397	proliferous	473, 487 499
Alteration in structure	4	squamous cell	455, 463, 465
Amyloid ..	35, 43		467, 471, 473,
Angiodermatoloma	378		475, 477 479
Angiodermatoloma—A. pom-		in	
mucosa	399 401	necrosis of the corium	.. 381
Angiolipoma	377 391	Ca. erous angioma	377 387
Angioma capillary	377 381 383, 385	Cell nest in basal squamous	
ca. erous	377 387	carcinoma	.. 481
hyperplastic	377 389	squamous cell	
Apoecrine sweat gland	13	carcinoma	463
sweat gland, active phase	17	Chancro—primary	51
duct	13	Cherodermatosis	123, 137
reticulate stage	15	Chondroma	419, 429
Atrophy	35	Circumflex keratoma	73
		Cle. m.	221
Bacillus lepro	291	Coil gland	8
Ballooning	3	Comedo in acne vulgaris	193
Balloon cells in nevi	169	Cordyloma squamatum	470, 469
Basal cells	7	latens	261
Basal-cell carcinoma	422 458, 467	Contagious erythema	.. 470
cystic type	459, 461	Copied ink deposit in skin	217
in nevus		Corn	210, 221
sebaceous	259	Cornu Rodri. in Darier disease	305
Basal squamous carcinoma	422, 461	Cutaneous horn	422, 463
Beil bag	73	Cutis lata	35, 41
Bleeder prurigo	102, 110, 123	Cystitis	213
Blue nevus	276, 231 233	Cyst—implantation	413, 415
Blue in pre cancerous dermatoses	423	ruleum	409 411
Buller in—keratodermatosis	137	sebaceous	.. 417
dermatitis herpetiformis	189	in haemorrhage	466 467
erythema multiforme	167	Cyst—acquired	407
hyperkeratosis	163	origin of	409
infectious contagiosa	177 179		
keratosis follicularis	87	Darier disease	274 303 305
keratosis palmis	161	Degeneration	35
keratosis palmis et plantarum	163	Dermatocystitis anularis	185
		Dermatitis herpetiformis	14 169
		Dermatocystoma	35, 36, 38
		Dermis ..	7
		Diffract papular eruption	102
		Dorothy Reed cells	.. 221
		Dysidrosis	135
		Dyskeratosis	.. 4
		Dyskeratotic cell in Darier	
		disease	.. 303
		Dystrophia	.. 38
		Eczema	107
		Bleeder prurigo	123

INDEX

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INDEX

[illegible]

	Page
Reticularis	20
Reticulo carcinoma	10
Reticulum an cutaneous carcinoma	243, 249
lymphadenoma	233
mycosis fungoides	237
Rhinophyma	201, 206
Rhinocleroma	33, 201
Rosacea	203, 208
Rosel flea	198
fly	223
Sarcoid	1, 277
Sarcoma—reticulo	223
large spindle-cell type	243, 248
test of sci	247, 249
Sarcophaga scabra	172, 178
Scabies	171, 172, 178
burrows	178
Scar	210, 228
Scleroderma	33, 48, 47
Sclerous at	487
gland	5, 23
duct	21
glands in scleroma	397
ulcerous	283, 287
necrosis	286
rhinophyma	205
rosacea	402, 408
retention cyst	311
Sclerobiosis	141, 183
Sclerous under epidermis	8
Sclerous in cutis	3
Sclerous follicles	189, 191
Sclerous cornu	8
fungus	7
granularis	7
granularis	3, 7
lucida	3, 7
Scleroma mult. plur.	223
Scleroma scleroma	40
Scleroma of	81
Scleroma	11, 227
Scleroma gland	2, 7, 9, 11
procur	13
scler	17
scler	16
scler	7, 9, 11
scler	358
scler	387, 389, 371, 373
scler	187
scler	201
scler	287, 289
scler	283, 285
scler	291
scler	211, 213

INDEX

	Page		Page
Tick	187	Verru—contagious	110
Tinea	199	Verru—contagious	111
corpora	201	Verru—contagious	112
kerion	201	Verru—contagious	113
Toutouguant II	435, 437	Verru—contagious	114
Traumatic injuries	409	Verru—contagious	115
Treponema pallidum	289	Verru—contagious	116
Tinea I le	278	Verru—contagious	117
Tumours	419	Verru—contagious	118
Ulna—nerve	10, 219	Verru—contagious	119
Ulna	61 65	Verru—contagious	120
papulosa	61 67	Verru—contagious	121
perforans	61 69	Verru—contagious	122
Urticaria	140, 181	Verru—contagious	123
—perforans	183	Verru—contagious	124
Urticaria	140	Verru—contagious	125
papulosa	147	Verru—contagious	126
—perforans	149	Verru—contagious	127
Urticaria	209, 219	Verru—contagious	128
Urticaria	140	Verru—contagious	129
Urticaria	8, 33	Verru—contagious	130
Urticaria	440, 443	Verru—contagious	131
Urticaria	440, 443	Verru—contagious	132
Urticaria	440, 443	Verru—contagious	133
Urticaria	440, 443	Verru—contagious	134
Urticaria	440, 443	Verru—contagious	135
Urticaria	440, 443	Verru—contagious	136
Urticaria	440, 443	Verru—contagious	137
Urticaria	440, 443	Verru—contagious	138
Urticaria	440, 443	Verru—contagious	139
Urticaria	440, 443	Verru—contagious	140
Urticaria	440, 443	Verru—contagious	141
Urticaria	440, 443	Verru—contagious	142
Urticaria	440, 443	Verru—contagious	143
Urticaria	440, 443	Verru—contagious	144
Urticaria	440, 443	Verru—contagious	145
Urticaria	440, 443	Verru—contagious	146
Urticaria	440, 443	Verru—contagious	147
Urticaria	440, 443	Verru—contagious	148
Urticaria	440, 443	Verru—contagious	149
Urticaria	440, 443	Verru—contagious	150
Urticaria	440, 443	Verru—contagious	151
Urticaria	440, 443	Verru—contagious	152
Urticaria	440, 443	Verru—contagious	153
Urticaria	440, 443	Verru—contagious	154
Urticaria	440, 443	Verru—contagious	155
Urticaria	440, 443	Verru—contagious	156
Urticaria	440, 443	Verru—contagious	157
Urticaria	440, 443	Verru—contagious	158
Urticaria	440, 443	Verru—contagious	159
Urticaria	440, 443	Verru—contagious	160
Urticaria	440, 443	Verru—contagious	161
Urticaria	440, 443	Verru—contagious	162
Urticaria	440, 443	Verru—contagious	163
Urticaria	440, 443	Verru—contagious	164
Urticaria	440, 443	Verru—contagious	165
Urticaria	440, 443	Verru—contagious	166
Urticaria	440, 443	Verru—contagious	167
Urticaria	440, 443	Verru—contagious	168
Urticaria	440, 443	Verru—contagious	169
Urticaria	440, 443	Verru—contagious	170
Urticaria	440, 443	Verru—contagious	171
Urticaria	440, 443	Verru—contagious	172
Urticaria	440, 443	Verru—contagious	173
Urticaria	440, 443	Verru—contagious	174
Urticaria	440, 443	Verru—contagious	175
Urticaria	440, 443	Verru—contagious	176
Urticaria	440, 443	Verru—contagious	177
Urticaria	440, 443	Verru—contagious	178
Urticaria	440, 443	Verru—contagious	179
Urticaria	440, 443	Verru—contagious	180
Urticaria	440, 443	Verru—contagious	181
Urticaria	440, 443	Verru—contagious	182
Urticaria	440, 443	Verru—contagious	183
Urticaria	440, 443	Verru—contagious	184
Urticaria	440, 443	Verru—contagious	185
Urticaria	440, 443	Verru—contagious	186
Urticaria	440, 443	Verru—contagious	187
Urticaria	440, 443	Verru—contagious	188
Urticaria	440, 443	Verru—contagious	189
Urticaria	440, 443	Verru—contagious	190
Urticaria	440, 443	Verru—contagious	191
Urticaria	440, 443	Verru—contagious	192
Urticaria	440, 443	Verru—contagious	193
Urticaria	440, 443	Verru—contagious	194
Urticaria	440, 443	Verru—contagious	195
Urticaria	440, 443	Verru—contagious	196
Urticaria	440, 443	Verru—contagious	197
Urticaria	440, 443	Verru—contagious	198
Urticaria	440, 443	Verru—contagious	199
Urticaria	440, 443	Verru—contagious	200

